HE 20.3313 : 9/9

GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM, AND DIGESTIVE DISEASES

RECEIVED
MIAMI UNIVERSITY
LIBRARIES
MAR 2 1976
DEPOSITORY
DOCUMENTS DEPARTMENT

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institutes of Health

SEPTEMBER 1975
VOLUME 9, NO. 9
3785—5196

Volume 9 No. 9 Abstract and Citation Nos. 3785–5196

GASTROENTEROLOGY ABSTRACTS AND CITATIONS

A monthly publication of the
National Institute of Arthritis, Metabolism, and Digestive Diseases

Editor

O. Dhodanand Kowlessar, M.D., Philadelphia

Associate Editors

Gordon D. Benson, M.D., Philadelphia
S. Philip Bralow, M.D., Philadelphia

Editorial Advisors

Thomas P. Almy, M.D., Hanover, New Hampshire
Frank P. Brooks, M.D., Philadelphia
Wilfrid I. Card, M.D., Glasgow
Charles F. Code, M.D., Rochester, Minnesota
Morton I. Grossman, M.D., Los Angeles
J. N. Hunt, M.D., London
Franz J. Ingelfinger, M.D., Boston
Joseph B. Kirsner, M.D., Chicago
Leonard Laster, M.D., Brooklyn, New York
Milton J. Matzner, M.D., Brooklyn, New York
Marvin H. Sleisenger, M.D., San Francisco
Borje Uvnas, M.D., Stockholm

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section
Bruce H. Kleinstein, Ph.D., Technical Editor
for the
National Institute of Arthritis, Metabolism, and Digestive Diseases
pursuant to Contract Number NO1-AM-4-2204
with the PHS, USDHEW

DHEW Publication No. (NIH) 76-66

PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

GASTROENTEROLOGY ABSTRACTS and CITATIONS is published monthly and includes citations and abstracts from the biomedical world literature as they are currently received. In addition, yearly cumulated subject and author indexes are published.

This publication is available free to National Institutes of Health grantees and contractors working in the field of gastroenterology and libraries of medical schools. Requests from these qualified individuals to receive free copies of this publication must include their grant or contract number and the title of their project. These requests as well as address changes and other communications should be addressed to:

Scientific Communications Office
Gastroenterology Abstracts and Citations
National Institute of Arthritis, Metabolism and Digestive Diseases
National Institutes of Health
Building 31, Room 9A-03
Bethesda, Maryland 20014

Other individuals and libraries may receive this publication by subscribing *directly* to the Superintendent of Documents, U.S. Government Printing Office, Washington D.C. 20402. Subscription price per year in the United States, Canada, and Mexico, $35.35 (this includes index issue); foreign, $8.85 additional. Single copies are $2.55, and the index issue is $4.75. Payment is required in advance, and check or money order should be made payable to the Superintendent of Documents.

CONTENTS

	Abstracts, Citations	Page
PRE-CLINICAL SCIENCES		
Morphology, Cellular Structure and Histochemistry	3785-3805	503
Absorption--Excretion--Transport	3806-3860	506
Motility	3861-3910	512
Secretion and Metabolism	3911-3919	519
Stomach	3920-3973	520
Pancreas	3974-3993	528
Hepatobiliary Tract	3994-4148	532
Intestine	4149-4163	545
General	4164-4185	547
DIAGNOSTIC PROCEDURES	4186-4298	551
GASTROINTESTINAL DISEASES		
Esophagus	4299-4365	560
Stomach	4366-4481	564
Peptic Ulcers	4482-4539	575
Small Intestine	4540-4601	585
Malabsorption	4602-4644	589
Large Intestine	4645-4708	594
Ulcerative Colitis	4709-4721	600
Pancreas	4722-4736	602
Pancreatitis	4737-4779	603
Liver and Biliary Tract	4780-4853	607
Hyperbilirubinemic States	4854-4856	616
Nonviral Hepatitis and Hepatic Injury	4857-4869	616
Acute Viral Hepatitis	4870-4898	619
Chronic Hepatitis	4899-4911	622
Alcoholic Liver Disease	4912-4917	624
Cirrhosis	4918-4939	625
Gallbladder and Biliary Tract Diseases	4940-5012	628
General	5013-5130	636
Parasitology	5131-5158	643
Regional Enteritis	5159-5196	645
AUTHOR INDEX		
SUBJECT INDEX		

The Secretary of the Department of Health, Education, and Welfare has determined that the publication of this periodical is necessary in the transaction of the public business required by law of this Department. Use of funds for printing this periodical has been approved by the Director of the Office of Management and Budget through April 30, 1975.

For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. Subscription price per year in the United States, Canada, and Mexico, $35.35 (this includes index issue); foreign, $8.85 additional. Single copies are $2.55, and the index issue is $4.75. Payment is required in advance, and check or money order should be made payable to the Superintendent of Documents.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mm	millimeter(s)
ADP	adenosine diphosphate	mOsm	milliosmolar; milliosmeter(s)
AMP	adenosine monophosphate	msec	millisecond(s)
amp	ampere	mV	millivolt(s)
ATP	adenosine triphosphate	min	minute(s)
Å	angstrom	MTD	maximum tolerated dose
BSP	sulfobromophthalein	NAD	nicotanamide adenine dinucleotide
C	degrees centigrade	NADH	reduced nicotanamide adenine dinucleotide
cal	calories	NADP	nicotanamide adenine dinucleotidephosphate
cc	cubic centimeter	NAPH	reduced nicotanamide adenine dinucleotidephosphate
cm	centimeter(s)	ng	nanogram (10^{-9})
dl	deciliter	nm	nanometer(s)
DNA	deoxyribonucleic acid	Pa	ambient pressure
ED_{50}	median effective dose(s)	PAS	periodic acid — Schiff
EDTA	ethylenediamine tetraacetic acid	pg	picogram (10^{-12})
e.g.	for example	pgEq	picogram equivalent(s)
EEG	electroencephalogram	p.o.	orally
ESR	erythrocyte sedimentation rate	ppb	parts per billion
g	gram(s)	ppm	parts per million
µg	microgram(s)	r	Roentgen
Hz	Herz	RBC	red blood cells (erythrocytes), red blood count
hr	hour(s)	resp.	respectively
i.m.	intramuscular	RNA	ribonucleic acid
i.p.	intraperitoneal	s.c.	subcutaneous
IU	international unit(s)	sec	second(s)
i.v.	intravenous	SGOT	serum glutamic-oxalacetic transaminase
kcal	kilocalorie(s)	SGPT	serum glutamic-pyruvic transaminase
kg	kilogram(s)	U	unit(s)
K_m	Michaelis constant	UV	ultraviolet
LD_{50}	median lethal dose(s)	V	volt
LDH	lactic acid dehydrogenase	WBC	white blood cells (leukocytes), white blood count
m	meter(s)	yr	year(s)
M	molar; mole(s)		
mM	millimolar; millimole(s)		
µM	micromolar; micromole(s)		
ma	milliampere(s)		
mCi, µCi	milli-, microcurie(s)		
mEq	milliequivalent(s)		
mg	milligram(s)		
ml, µl	milli-, microliter(s)		

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

3785 GROWTH-HORMONE RELEASE-INHIBITING HORMONE IN GASTROINTESTINAL AND PANCREATIC D CELLS. *(E.)* Polak, J. M.; Grimelius, L.; Pearse, A. G. E.; Bloom, S. R.; Arimura, A. (R. Postgrad. Med. Sch., London, England). *Lancet* 1(7918):1220-1222, 1975.

To determine whether growth-hormone release-inhibiting hormone (GH-RIH) is present in endocrine cells outside of the hypothalamus, the upper gastrointestinal mucosa and pancreatic D cells of dogs, pigs, and humans were examined for hormone activity using combined immunocytochemical and histological methods. Cells specifically stained for GH-RIH were seen in all areas of the gastrointestinal mucosa examined, being predominantly localized in the midzone of the glands. The hormone-positive cells were lead-hematoxylin positive but negative with the Masson and Grimelius silver-impregnant techniques. GH-RIH cells were also seen in the human pancreatic islets; these cells corresponded to the argyrophil cells stained with the Hellerstrom-Hellman method for D (α_1) cells. The GH-RIH cells were negative for the Grimelius silver method, which stains the glucagon-producing A cells of the pancreas positively. The results suggest that in the gut, the D cell of the Wiesbaden classification is the most likely source of GH-RIH. They also suggest that GH-RIH may play an important role in digestive regulation.

3786 MORPHOLOGICAL INCONSISTENCY IN LIVER CELLS IN CULTURE. *(E.)* Okigaki, T.; Quan, S. G. (Pasadena Found Med. Res., Calif.). *Proc. Jpn. Acad.* 50(7):517-520, 1974.

Morphological changes were studied in cultured rat hepatocytes. Livers from three 12-day-old Wistar rats were minced and dissociated with a mixture containing collagenase, hyaluronidase, and trypsin. A primary culture, grown for 2 weeks in Ham's F-12 medium with 10% fetal calf serum, showed a mixture of hepatocyte-like epithelial and non-epithelial cells. The non-epithelial cells were mechanically eliminated with a sharp pin, and a homogenous population of epithelial cells obtained by clonal isolation and/or treatment with hydrocortisone (20 mg/liter) containing medium. The cultures were studied by time lapse cinematography. Typical epithelial cells in early passages were observed to change their morphology in a relatively short period ot time. One line was not epithelial in morphology in later passages, and showed no liver specific response for tyrosine transaminase, but showed a weak positive reaction for tyrosine aminotransferase. This line had been grown in both arginine-free and hydrocortisone-containing media. Although lacking certain liver specific characteristics, it reacted to thioacetamide in the same manner as normal hepatocytes, with swelling and deformation of nucleoli. It is concluded that cultured liver cells can be classified as typical epithelial cells with liver specific characteristics, hepatocyte-derived near- or non-epithelial cells not retaining many characteristics, and non-epithelial non-hepatocytic cells from the beginning of cultivation.

3787 ULTRASTRUCTURE OF THE DEVELOPING FETAL HEPATOCYTE OF Macaca arctoides: A PROPOSED MODEL FOR STUDIES OF FETAL DRUG METABOLISM. *(E.)* Quattropani, S. L.; Stenger, V. G.; Dvorchik, B. H. (Milton S. Hershey Med. Cent., Pennsylvania State Univ., Hershey). *Anat. Rec.* 182(1):103-122, 1975.

The ultrastructure of various developmental stages of the fetal hepatocyte was studied in the stumptail monkey *(Macaca arctoides)*. Groups of six to eight females were mated, and pregnancy and gestational age were determined by bimonthly rectal palpations of the uterus. Fetuses were delivered by Caesarian section between 8:15 and 8:45 A.M. at either 80 (midterm) or 160 (nearterm) days gestation. Immediately following delivery, a slice of the anterior medial portion of the right hepatic lobe was taken from the fetus, fixed in glutaraldehyde, and postfixed in osmium tetroxide. Thin sections were stained in uranyl acetate and lead citrate and studied by electron microscopy. Thick sections of the same blocks (0.5-1.0 μ) were stained with methylene blue and azure C and examined by light microscope. Equivalent specimens were also obtained from a 1-week-old monkey and 12 and 16 week human fetuses. At midterm, 50-70% of a given field was hepatocytes, the rest being hemopoietic tissue. The amounts of hemopoietic tissue and smooth endoplasmic reticulum varied with age, the former decreasing and the latter increasing. The smooth endoplasmic reticular elements were concentrated at the edges of the rough endoplasmic reticular stacks in the immature nearterm. By full nearterm there was a pronounced aggregation of smooth elements in the peripheral cytoplasm. Similar aggregates were seen at 1 week postnatal age. It is concluded that fetal monkey hepatocytes have smooth endoplasmic reticulum, which correlates with their ability to metabolize drugs.

3788 ULTRASTRUCTURAL AND CYTOCHEMICAL STUDY OF MUCOUS GRANULES IN SURFACE AND CRYPT CELLS OF RAT GASTRIC MUCOSA. *(E.)* Yeomans, N. D. (Anat. Dep. C, Univ. Copenhagen, Denmark). *Biol. Gastroenterol.* 7(4):285-290, 1974.

The composite mucous granules in gastric mucosa surface and crypt cells were studied for frequency of occurrence at particular sites, reactivity for complex carbohydrates, and susceptibility to proteolytic digestion. Tissues from antral and fundic mucosa from 10 Wistar rats were fixed by perfusion with glutaraldehyde. Some were postfixed with osmium tetroxide. The mucosal surface and upper quarter of the crypts and the bottom of the crypts were examined by electron microscopy and 1000 granules counted in each

region. Glycoproteins were demonstrated by a modification of the periodic-chromic acid-silver methenamine method, and protein digestion was carried out by incubation with pronase E. Two types of mucous granule profiles were seen in the surface and crypt cells, depending on the method of fixation. When postfixed with osmium, some granules appeared moderately electron dense and homogenous; the rest appeared to have a highly electron dense core surrounded by a moderately dense outer shell. The cores were central or eccentric with a modal diameter of 250 nm; core to granule diameter ratio was about 1:2. When fixed with glutaraldehyde only, the homogenous granules and outer shells of the composite granules appeared obviously extracted. Granules with cores were more common at the mucosal surface and upper parts of the crypts than at the crypt bottoms both in the antrum and in the fundus. The homogenous granules and outer shells of the composite granules gave a strong cytochemical carbohydrate reaction, but the composite granule cores were non-reactive. Proteolytic digestion removed the cores while leaving the outer shells unchanged. It is concluded that composite granules are common at the surface of rat gastric mucosa and contain cores which are probably of protein with little or no carbohydrate.

3789 ULTRASTRUCTURAL LOCALIZATION OF ACTIN-LIKE FILAMENTS IN RAT HEPATOCYTES. *(E.)* French, S. W.; Davies, P. L. (Dep. Pathol., Univ. British Columbia, Vancouver, Canada). *Gastroenterology* 68(4): 765-774, 1975.

The heavy meromyosin binding technique was used to ultrastructurally identify actin-like microfilaments in hepatocytes. Liver and parotid tissue from three Wistar rats was glycerinated and incubated with heavy meromyosin in three experiments. Pieces of tissue (1-2 mm cubes) were placed in standard salt solution containing 50% glycerol. The glycerol concentration was reduced to 25% and after 4-12 hr was replaced by heavy meromyosin for 24 hr. After fixation in glutaraldehyde, tissues were examined by electron microscope. The parotid tissue served as a positive control. Negative controls were not exposed to heavy meromyosin. The effects of ATP and pyrophosphate on heavy meromyosin binding to actin filaments were tested in two experiments by including these substances with the heavy meromyosin during the filament decoration step. Results were similar for both liver and parotid tissues. In glycerated cells not incubated with heavy meromyosin two filament types were observed: a fine interconnecting network of filaments 5.4-5.9 nm in diameter adjacent to the plasma membrane; and straight filaments, single or in bundles, parallel to the plasma membrane and also deep within the cytoplasm. In the heavy meromyosin-incubated liver, the fine filament network appeared much more abundant than in controls; individual filaments were thicker and covered with spikelike extensions. They connected with the plasma membrane, organelles, and each other, and intersected at close to 90° angles. The straight filaments, in contrast, did not bind heavy meromyosin and appeared to be reduced in number after incubation. They ranged from 8-10 nm in diameter. Pyrophosphate inhibited heavy meromyosin decoration incompletely, while ATP caused complete inhibition. The filament network was largely beneath the plasma membrane facing the sinusoid but was also prominent around the bile canaliculus; the distribution corresponded to the polygonal pattern of actin, as demonstrated by smooth muscle binding antibodies. It is concluded that the ectoplasmic microfilament network is actin and is part of the cell's contractile system.

3790 ONTOGENETIC CHANGES IN THE ULTRASTRUCTURE OF RAT HEPATOCYTE ORGANELLES AFTER PRENATAL X-IRRADIATION. *(E.)* Chedid, A.; Nair, V. (Univ. Cincinnati Sch. Med., Ohio). *Radiat. Res.* 62(1):123-132, 1975.

The radiation-induced suppressive effect on enzyme development in the rat hepatocyte was studied with respect to ultrastructural changes in the endoplasmic reticulum. Pregnant Sprague-Dawley rats received 50 r to the pelvic region on the 13th gestation day and were sacrificed on the 15th and 20th gestation days. In a postnatal series, offspring were sacrificed on the day of birth and the fifth postnatal day. Liver specimens from four fetuses or neonates of each time period were cut into 1 mm^3 cubes and examined by electron microscopy. On the 15th gestation day, fetal hepatocytes, in addition to the normal even cytoplasmic distribution of ribosomes, showed large dense polyribosomal aggregates near the nucleus and cell periphery, sometimes with numerous microtubules and dilatation of the rough endoplasmic reticulum. In the 20th gestation day hepatocytes, smooth endoplasmic reticulum failed to appear. By the day of birth, the polyribosomal aggregates were gone, but smooth endoplasmic reticulum still had not appeared, and necrotic hepatocytes were common in the centrilobular area. In the fifth postnatal day the smooth endoplasmic reticulum had appeared. It is concluded that in conjunction with previous findings, lipid peroxidation, membrane alteration, delayed appearance of smooth endoplasmic reticulum, and polyribosomal aggregation may be sequentially linked events after irradiation.

3791 ELECTRON MICROSCOPE AUTORADIOGRAPHIC STUDY OF INTESTINAL ABSORPTION OF DECANOIC AND OCTANOIC ACIDS IN THE RAT. *(E.)* Carlier, H.; Bezard, J. (Inst. Appl. Biol. Nutr. Aliment., Dijon, France). *J. Cell Biol.* 65(2):383-397, 1975.

Intestinal absorption of [^{3}H]octanoic acid and [^{3}H] decanoic acid was investigated in the rat by electron microscope autoradiography. The common duct of the rats was diverted and a loop of the duodenum was cannulated 24 hr later. Tritiated octanoic acid and decanoic acid were introduced into the lumen of each duodenal loop, and uptake was allowed to proceed for various times up to 15 min. The loop was then removed; one part was used to determine the distribution of radioactivity in different lipid fractions, and an autoradiographic study was performed on the other part of the loop. Radioactivity distribution showed that medium chain fatty acids were absorbed

in their nonesterified form and that they were absorbed much more rapidly than oleic acid, a long chain fatty acid. Autoradiographic studies indicated that the medium chain fatty acids were taken up in a molecular or aggregate molecular form, left the epithelial cells by way of the lateral plasma membrane, and were next found in the blood capillaries. The results suggest that the Golgi complex does not play an important role in the absorption of unesterified fatty acids, but seems involved only in the transfer of esterified long chain fatty acids.

3792 HISTOLOGICAL CHANGES IN THE INTESTINAL TRACT OF PREGNANT MICE INFECTED WITH COXSACKIE-VIRUS B3. *(E.)* Lansdown, A. B. G. (Clin. Res. Cent., Harrow, England). *Experientia* 31(3):327-328, 1975.

The possibility that protein malnutrition in mice infected with coxsackievirus B3 may be sufficient to elicit morphological changes in the intestinal tract leading to an impaired absorption of essential nutrients which are important for normal fetal growth, as well as for maintaining good maternal health, was examined. Mice were inoculated i.m. with 0.3 ml live or heat-inactivated virus suspension on the eighth day of pregnancy and killed by cervical dislocation on the 18th day. Representative segments of liver, pancreas, stomach, duodenum, ileum, cecum, and colon were fixed in phosphate buffered formalin for histological examination. The stomach and upper regions of the intestinal tract of infected animals appeared normal. The ileum, cecum, and upper colon, however, appeared to be dilated and edema of the lamina propria was identified. In the cecum and colon profound edema was present with a marked dilatation of the gut lumen associated with a thinning and distension of the mucosae. Focally the mucosa was reduced to a single layer of cuboidal cells with few mucus-secreting cells and poorly defined basement membrane. The intestinal changes of edema and focal mucosal atrophy are consistent with a short period of protein deficiency. The implications of these intestinal changes upon the state of health of the pregnant mouse or on the development of the fetuses at 18 days gestation are unclear. In view of previously published evidence that the absorptive capacity of the gut is altered only in advanced cases of protein deficiency resulting from several months deprivation, it is unlikely that the early intestinal changes present in these coxsackievirus-infected mice at the stage examined present any appreciable risk to the health of pregnant mice.

3793 THE ULTRASTRUCTURE OF BRUSH BORDER CELLS IN THE INTESTINE OF NINE TO TEN-WEEK-OLD HUMAN EMBRYOS. *(Rus.)* Kovalchuk, V. K. (Kiev Sci. Res. Inst. Infect. Dis., USSR). *Arkh. Anat. Gistol. Embriol.* 67(10):74-77, 1974.

Electron microscope examination of the intestinal mucosa from 9 to 10 week old human embryos revealed a wide range of intracellular differentiation. Deep inside the mucous gland, brush border cells were poorly differentiated: there were few mitochondria with isolated cristae and poorly developed endoplasm and Golgi's cells. The central regions of the supranuclear zone contained finely granular electron-transparent material which looked like the cytoplasmic matrix of continuously cultured cells. As the differentiation proceeded, this material was replaced by the membranes of the endoplasmic system, ribosomes and mitochondria. In the overlying parts of the crypts, there was dense granular cell structures amidst the cytoplasmic structures. The poor differentiation of brush border cells was combined with enzyme deficiency which was manifested by the negative reaction to acid phosphatase. These findings suggest that the surface layer of the intestinal epithelium may develop during early histogenesis and undergo molecular rearrangement after birth.

3794 FUNCTIONAL POLARITY OF THE RAT HEPATOCYTE SURFACE MEMBRANE: ISOLATION AND CHARACTERIZATION OF PLASMA-MEMBRANE SUBFRACTIONS FROM THE BLOOD-SINUSOIDAL, BILE-CANALICULAR AND CONTIGUOUS SURFACES OF THE HEPATOCYTE. *(E.)* Wisher, M. H.; Evans, W. H. (Natl. Inst. Med. Res., London, England). *Biochem. J.* 146(2):375-388, 1975.

The distribution of a hormone-sensitive adenylate cyclase and a range of enzymic, chemical, and morphological properties were examined in 6 purified plasma-membrane subfractions of liver homogenates from fasted Sprague-Dawley rats. The six subfractions were prepared using a combination of differential, rate-zonal, and density-gradient centrifugation. There were 3 vesicular 'light' subfractions of density 1.12-1.13 and 3 'heavy' subfractions of density 1.16-1.18 containing membrane strips and intercellular junctions. All the subfractions contained a basal adenylate cyclase activity. In one of the light subfractions, the highest glucagon-stimulated adenylate cyclase activity was derived from the blood-sinusoidal face of the hepatocyte; unlike the others, this subfraction was contaminated by Golgi components. All of the subfractions also showed high activities of the following plasma-membrane marker enzymes: 5'-nucleotidase, alkaline phosphodiesterase (nucleotide pyrophosphatase), alkaline phosphatase, leucine naphthyl-amidase, and Mg^{2+}-activated ATP. One light subfraction which showed the highest specific activities of these marker enzymes but lacked a glucagon-stimulated adenylate cyclase activity derived from the bile-canalicular face of the hepatocyte. The heavy subfractions generally showed the lowest enzyme activities, were characterized by the presence of desmosomes and gap junctions, and appeared to originate from the continguous faces of the hepatocyte. Polyacrylamide-gel electrophoresis indicated a general similarity in the protein compositions of the 6 subfractions. Differences in the amounts of various protein and glycoprotein bands correlated with differences in morphology, enzymic composition, and sialic acid content. These results, in conjunction with previous plasma-membrane hormone-binding and histochemical data, indicate the existence of functionally discrete plasma-membrane subfractions originating from geographically separated regions of the hepatocyte surface.

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

3795 SEBACEOUS GLANDS IN THE WALL OF THE ANAL CANAL OF RATS, MICE AND CLOSELY RELATED RODENTS. *(E.)* Shehata, R. (Fac. Med., Cairo Univ., Egypt). *Acta Anat. (Basel)* 90(2):243-249, 1974.

3796 AN ELECTRON MICROSCOPIC STUDY OF THE PANCREAS AND PAROTID GLAND OF RATS WITH EXPERIMENTAL ACUTE PANCREATITIS. *(E.)* Ishidate, T.; Senoo, A.; Kakizaki, G.; Saito, T.; Fujiwara, Y.; Nihei, T. (Akita Univ. Sch. Med., Japan). *Tohoku J. Exp. Med.* 113(3):213-223, 1974.

3797 THE ULTRASTRUCTURAL LOCATION OF ARYLSULFATASE IN LYSOSOMES. *(Rus.)* Pokrovskii, A. A.; Krystev, L. P.; Tutelian, V. A.; Kravchenko, L. V. (Inst. Nutr., Moscow, USSR). *Dokl. Akad. Nauk SSSR* 222(3):713-715, 1975.

3798 TECHNIQUE FOR STUDYING THE DISTRIBUTION OF GASTRIN CELLS IN THE NORMAL AND DISEASED HUMAN STOMACH. *(Fr.)* Potet, F.; Constantin-Voillemot, N.; Lewin, M. (Beaujon Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4):296, 1974.

3799 GASTRIN CELLS IN THE STOMACH. *(Fr.)* Potet, F.; Constantin-Voillemot, N. (Beaujon Hosp., Clichy, France). *Ann. Anat. Pathol. (Paris)* 19(4):400-412, 1974.

3800 MORPHOLOGY OF REABSORBING GASTROINTESTINAL EPITHELIA. *(Ger.)* Kulenkampff, H. (Dep. Anat., Univ. Saarland, Homburg/Saar, Germany). *Arzneim. Forsch.* 25(3):461-463, 1975.

3801 AUTORADIOGRAPHIC ANALYSIS OF CELL POPULATION KINETICS IN THE SMALL INTESTINE AND COLON EPITHELIUM DURING EARLY POSTNATAL DEVELOPMENT. *(Rus.)* Zufarov, K. A.; Lomonosova, G. A.; Irgashev, S. B.; Yuldashev, A. Y. (Med. Inst., Tashkent, USSR). *Cytologia (Mosk.)* 16(9):1106-1110, 1974.

3802 AUTORADIOGRAPHIC STUDY OF CELL POPULATION KINETICS IN THE INTESTINAL AND LIVER EPITHELIUM OF Rana temporaria. *(Rus.)* Lanskaya, A. B. (Dep. Cytol. Histol., Leningrad Univ., USSR). *Cytologia (Mosk.)* 16(3):329-334, 1974.

3803 HISTOCHEMISTRY, IMMUNOFLUORESCENCE, AND MODERN PROCEDURES FOR STUDYING RECTAL BIOPSIES. *(Fr.)* Laumonier, R.; Huitric, E.; Delpech, A. (Henri Becquerel Cent., Rouen, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(4):315-320, 1974.

3804 THE EFFECTS OF COSMIC PARTICLE RADIATION ON PACKET MICE ABOARD APOLLO XVII: APPENDIX III: EVALUATION OF VISCERA AND OTHER TISSUES. *(E.)* Ellis, J. T.; Kraft, L. M.; Lushbaugh, C. C.; Humason, G. L. Hartroft, W. S.; Porta, E. A.; Bailey, O. T.; Greep, R. O.; Leach, C. S.; Laird, T.; Simmonds, R. C.; Vogel, F. S.; Dennis, R. L.; Brashear, H. R.; Talmage, R. V.; Harrison, G. A.; Corbett, R. L.; Klein, G.; Tilbury, T.; Suri, K.; Haymaker, W. (New York Hosp.-Cornell Univ. Med. Cent., N.Y.). *Aviation Space Environ. Med.* 46(4):639-654, 1975.

3805 VASCULAR ALTERATIONS IN CANINE HISTIOCYTIC ULCERATIVE COLITIS. *(E.)* Lawson, T. L.; Gomez, J. A.; Margulis, A. R. (Univ. California Sch. Med., San Francisco). *Invest. Radiol.* 10(3):212-224, 1975.

See also, 3960, 4030, 4053, 4064, 4101, 4175, 4157, 4432, 4489, 4620, 5102.

ABSORPTION-EXCRETION-TRANSPORT

3806 FERRITIN IRON ABSORPTION IN MAN. *(E.)* Layrisse, M.; Martinez-Torres, C.; Renzy, M.; Leets, I. (Dep. Exp. Med., Venezuelan Inst. Sci. Invest., Caracas). *Blood* 45(5):689-698, 1975.

The effect of vegetable and animal foods on the iron absorption from ferritin was studied in 108 Venezuelan peasants. Following an overnight fast, the subjects ingested ferritin alone or mixed with various foods which had been biosynthetically tagged with radioiron. Blood was drawn 15 days later to determine hematologic characteristics and radioactivity. The geometric mean absorption of ferritin iron in both normal and iron-deficient subjects was 1.9%, the mean absorption ranging from 0.9% in the normal subjects to 2.5% in those with moderate iron deficiency to 5.7% in those with marked iron deficiency. The administration of ferritin with vegetable foods (maize, wheat, soybeans) resulted in lower absorption values than those obtained with the same foods alone. The iron absorption from ferritin was markedly increased when it was administered with meat or liver, but it did not reach the level observed with these foods alone. The mean iron absorption from 1 mg of ferritin iron was 2.3%, while that from 1 mg of ferric chloride was 11.7%. Carrier liver increased the absorption of both substances and carrier maize markedly reduced the absorption of ferric chloride. When ferritin was administered with 100 mg of ascorbic acid, the geometric mean absorption of iron was significantly increased over that seen with ferritin alone. The results indicate that purified ferritin

has a very low absorbability and that its absorption is affected by substances which enhance or reduce iron absorption in the same proportion as vegetal iron.

3807 DIFFERENT PATHWAYS FOR HEPATIC UPTAKE OF TAUROCHOLATE AND INDOCYANINE GREEN. *(E.)* Paumgartner, G.; Reichen, J. (Dep. Clin. Pharmacol., Univ. Berne, Switzerland). *Experientia* 31(3):265-392, 1975.

To determine whether different pathways exist for the hepatic uptake of bile acids and anionic dyes, the kinetics of the hepatic uptake of taurocholate and indocyanine green were compared in the perfused rat liver. The livers of specific-pathogen-free male Sprague-Dawley rats were perfused *in situ*, during which ^{14}C-labeled taurocholate (75-25,000 nM) and/or indocyanine green (ICG, 60-50,000 nM) was rapidly injected into the portal vein along with $^{99}Tc^{m}$-albumin. The total venous outflow was collected and the radioactivity counted. Taurocholate and albumin were distributed in equal spaces and a certain fraction of the taurocholate was removed by the liver. Similar dilution curves were obtained with ICG, which was also distributed in equal spaces with albumin. The initial uptake rate of both taurocholate and ICG increased with the extra-vascular dose in a nonlinear fashion, exhibiting saturation kinetics. Analysis of these data revealed compatibility with Michaelis-Menten kinetics for both anions. The maximal uptake capacity of taurocholate exceeded that of ICG by a factor of 9. The apparent half-saturation constants for the hepatic uptake of taurocholate and ICG were 90.6 and 50.0 nM/g liver, resp. There was no significant competition between taurocholate and ICG for uptake by the hepatocytes. The results suggest that different pathways exist for the transport of bile acids and anionic dyes from the blood into the hepatocyte.

3808 FURTHER STUDIES ON THE ACTION OF PROLACTIN ON FLUID AND ION ABSORPTION BY THE RAT JEJUNUM. *(E.)* Mainoya, J. R. (Dep. Zool., Univ. California, Berkeley). *Endocrinology* 96(5):1158-1164, 1975.

The effects of *in vivo* prolactin administration on the *in vitro* absorption of fluid and ions by the rat jejunum were studied. Ovine prolactin was administered s.c. to intact and hypophysectomized male Sprague-Dawley rats. Twenty-four or 48 hr later, the nonfasted animals were anesthetized and everted sacs were prepared from segments I-V of the small intestine. The sacs were filled with Ringer solution containing glucose and placed in metabolic incubators, after which mucosal transport was measured. Prolactin administration significantly stimulated fluid, sodium, potassium, calcium, magnesium, and chloride transport across the everted jejunal sacs, the latter 2 ions being the least affected. Hypophysectomy caused a significant decrease in fluid and sodium absorption which was prevented by prolactin treatment. The effects of prolactin on fluid and sodium absorption were dose-dependent, maximal stimulation resulting from the administration of 1.0-2.0 mg; higher doses failed to elicit a significant response. The stimulatory action of prolactin was inhibited by the simultaneous administration of vasopressin, which when given alone had no effect on intestinal absorption. In the absence of glucose or in the presence of phlorizin, fluid transport was inhibited, the reduction most dramatic in the presence of phlorizin. Fluid transport was also reduced by the application of ouabain or partial replacement of sodium with isotonic choline chloride. These results suggest that the effects of prolactin on intestinal transport may be dependent on the increased movement of sodium.

3809 KINETICS OF Na^+ UPTAKE AND TRANSCELLULAR TRANSIT BY THE PANCREAS. *(E.)* Rossier, M.; Rothman, S. S. (Dep. Physiol., Univ. California, San Francisco). *Am. J. Physiol.* 228(4):1199-1205, 1975.

The kinetics of Na^+ uptake and transcellular transit by pancreatic tissue was studied to determine the model for tissue uptake of the molecule. ^{22}Na uptake into strips of rabbit pancreas was measured for up to 10 min. The uptake curve was characterized by the presence of 2 plateaus separated by an inflexion point; a 1st plateau was observed between 1 and 3 min; between 3 and 4 min the slope of the uptake curve increased again; it finally decreased to a new and higher steady-state uptake between 4 and 6 min. The data suggest that the 1st part of the uptake curve (0-3 min) represents uptake into the cells, and the 2nd part (3-10 min) represents the sum of "quasi" steady-state cellular uptake and of the equilibration of the ductal compartment in series with the cells. A delay of 2.5-3.25 min elapsed between the filling of cellular and ductal compartments, which appeared to be of intracellular origin (restricted Na^+ diffusion within the cytoplasm and an intracellular Na^+ gradient). Based on that model, the transit time for Na^+ across the whole organ was expected to be 3-4 min and was found to be 3.5 min when studied *in vitro*. The transepithelial transit of Na^+ appeared to account for most of that time. The data indicate that a substantial delay exists between the filling of the cells and the ducts; thus, tissue uptake may be described as the sum of an exponential and a sigmoid function.

3810 EFFECT OF A COMBINED THIAMINE AND RIBOFLAVIN DEFICIENCY ON INTESTINAL ABSORPTION OF ESSENTIAL AMINO ACIDS. *(Fr.)* Wellers, G.; Chevan, J. (Unit. Inform. Biomed. Res., Paris, France). *Ann. Nutr. Alim.* 28(1):49-63, 1974.

Male Wistar rats were fed a diet containing all 10 essential amino acids plus other essential nutrients but deficient in thiamine and riboflavin. After 20 days, the rats were deprived of all amino acids, but glycine and L-glutamic acid were added to the diet. Three days later, the rats were fasted for 24 hr and a mixture containing all previous nutrients was instilled into their stomachs. After 6 hr, the animals were sacrificed and the

amino acid levels in the intestinal contents, blood and gastrointestinal tissues were determined by microbiological assays. Thiamine-deficient rats absorbed arginine at a normal rate, but the other nine essential amino acids were absorbed more slowly than normal. A riboflavin deficiency increased the absorption rate for all amino acids but tryptophan. When rats were deficient in both thiamine and riboflavin, the absorption of histamine, leucine, lysine, methionine, phenylalanine, threonine, and valine were decreased, while arginine, isoleucine, and tryptophan were absorbed at normal rates. Disorders in amino acid absorption in vitamin-deficient rats may be the primary cause of signs of disorders in protein metabolism (weight loss, anorexia) that were observed.

3811 LIVER UPTAKE OF AMINO ACIDS AND CARBOHYDRATES DURING A SINGLE CIRCULATORY PASSAGE. *(E.)* Pardridge, W. M.; Jefferson, L. S. (Milton S. Hershey Med. Cent., Hershey, Pa.). *Am. J. Physiol.* 228(4):1155-1161, 1975.

The uptake of ^{14}C-labeled amino acids and carbohydrates by rat liver following rapid injection into the portal vein was measured using tritiated water as an internal standard; the applicability of this technique (Oldendorf technique) to the study of liver transport systems was then judged. A 0.25 ml bolus of buffered Ringers solution containing 1-2 µCi of the ^{14}C-labeled test substance 3-6 µCi of tritium-labeled water (^{3}HOH) was administered by rapid portal injection into anesthetized male Sprague-Dawley rats. Circulation was terminated after a single passage of the bolus through the hepatic microvasculature and the tissue was immediately macerated, solubilized, and subjected to liquid scintillation counting. Liver uptake indices (LUI) were calculated from the ratio of ^{14}C to ^{3}H in the liver tissue relative to this ratio in the injection mixture. The LUI's of sucrose, inulin, D-mannitol, D-glucose, and L-glucose were 24.3%, 27.7%, 80.2%, 96.8%, and 26.7%, resp.; the LUI for cholic acid was 127.1%. Among the 18 amino acids tested, the LUI's were highest for the acidic compounds (e.g., L-aspartic acid, 100%, and L-glutamic acid, 86.4%) and lowest for the more basic compounds (e.g., L-arginine, 37.4%, and L-lysine, 31.4%). Stereospecificity for glucose and alanine uptake, saturation kinetics for glutamic acid (K_m = 4.8 mM) and aspartic acid (K_m = 2.7 mM), and cross-inhibition among uptake of the various amino acids were observed. The data confirm the applicability of the Oldendorf technique, which was originally developed for studies of amino acid uptake into the brain, for the characterization of transport systems in the liver.

3812 EFFECTS OF CHRONIC ETHANOL ADMINISTRATION ON INTESTINAL ABSORPTION IN MAN IN THE ABSENCE OF NUTRITIONAL DEFICIENCY. *(E.)* Lindenbaum, J.; Lieber, C. S. (Harlem Hosp. Cent., New York, N.Y.). *Ann. N.Y. Acad. Sci.* 252:228-234, 1975.

The effects of chronic alcohol administration on intestinal absorption in man was studied under controlled metabolic ward conditions to determine whether ethanol alone, in the absence of associated nutritional deficiency, causes impairment of small bowel function. Asymptomatic middle-aged male volunteers with histories of chronic alcoholism were given 173-253 g/day of ethanol (46-66% of the total caloric intake) along with vitamin supplementation. Vitamin B_{12} absorption was studied by the urinary excretion method of Schilling following the oral administration of ^{57}Co-cyanocobalamin (0.75 µg) with intrinsic factor concentrate. The excretion of urinary xylose and fecal fat were also determined following the oral or i.v. administration of D-xylose. In 6 subjects, the excretion of B_{12} was reduced during ethanol administration compared with periods of abstinence (control); no depression in vitamin B_{12} absorption was seen in the other 2 subjects, both of whom received the smallest amount of ethanol. The concomitant administration of pancreatic extract did not improve B_{12} absorption during ethanol administration. Urinary xylose and serum xylose increased during ethanol administration and fecal fat excretion was reduced. The absorption of xylose and fat was not inhibited by alcohol administration. The results indicate that chronic ethanol administration results in impaired B_{12} absorption in man despite adequate nutrient intake.

3813 PASSAGE OF PARTICLES THROUGH THE WALL OF THE GASTROINTESTINAL TRACT. *(E.)* Volkheimer, G. (no affil.). *Environ. Health Perspect.* 9:215-225, 1974.

The persorption of macrocorpuscular particles through the wall of the gastrointestinal tract is discussed in terms of mechanisms, rate, and the elimination, phagocytosis, enzymatic degradation, and deposition of persorbed particles. Persorption of particles in the micrometer size range is possible wherever the intestinal mucosa is covered by a single layer of epithelium. Primary transportation of persorbed particles from the digestive tract may take place in the chyle and *via* the portal circulation. The persorbed particles are found between the cells of the epithelial layer, in the subepithelial region of the mucosa, and within the lumen of lymph and blood vessels. The rate of persorption depends on factors such as the age of the subject, quantity of particles supplied, and the muscular activity of the intestine; the rate increases during deep physiological sleep and under the influence of cigarette smoking, caffeine, and prostigmine, and decreases during barbiturate sleep. Persorbed particles may be eliminated in the urine, bile, and milk (in lactating women), and have been observed to pass into the peritoneal cavity and cerebrospinal fluid; extravasation from the vessels in the pulmonary alveoli and transplacental passage have also been confirmed. Phagocytosis by micro- and macrophages was observed in the peritoneal cavity and spleen, and enzymatic degradation of starch granules in the body fluids was demonstrated. Finally, after long-term oral administration, deposition of embolized starch granules and other persorbed particles in the lumen of the smallest vessels has been noted.

3814 MEASUREMENT OF SUGAR ABSORPTION DURING DIGESTION OF CORNSTARCH IN SWINE. *(Fr.)* Rerat, A.; Aumaitre, A.; Vaissade, P.; Vaugelade, P. (Lab. Nutritional Physiol., INRA, Jouy-en-Josas, France). *C. R. Acad. Sci. [D] (Paris)* 279(10):831-834, 1974.

Glucose absorption during cornstarch digestion was measured quantitatively in 4 swine fitted with permanent cannulas in the portal vein and the anterior vena cava. Blood volumes were continuously recorded with electromagnetic flow meters. Experimental diets containing 150g protein, vitamins, minerals, and 400-1200g cornstarch were fed after 16 or 22 hr fasts, and blood glucose levels were monitored from 30 min before to 8 hr after meals. In spite of large variations in blood flow, the quantities of glucose absorbed were very consistent. Absolute quantities of glucose absorbed were directly proportional to the amount of cornstarch fed, but were relatively higher when smaller portions were ingested. The digestion time was very long and may be as long as 22-29 hr when large quantities of cornstarch are fed. This is in marked contrast to the absorption of amino acids after digestion of proteins, which is almost complete after 8 hr.

3815 THE INFLUENCE OF GLUCAGON ON JEJUNAL ABSORPTION OF GLUCOSE, WATER AND ELECTROLYTES IN MAN. *(Ger.)* Gottesburen, H.; Leising, H.; Menge, H.; Lorenz-Meyer, H.; Riecken, E. O. (Med. Clin., Univ. Marburg/Lahn, Germany). *Klin. Wochenschr.* 52(19):926-929, 1974.

The influence of glucagon on the resorption of glucose, water, sodium and potassium was studied by means of the segmental small-intestinal perfusion method with a three-channelled needle in the upper jejunum in three groups of subjects. These included healthy adults; eight maturity-onset diabetics with reduced insulin secretion after glucagon dose; and six patients with juvenile diabetes mellitus who required insulin. After measuring resorption over two 30-min periods the test subjects received 1 mg i.v. of glucagon and the resorption was determined during four 15 min periods. After glucagon administration in all three groups there was an increase of short duration in glucose absorption. This was more pronounced in the diabetics than in the healthy subjects. Subsequently a significant decrease of glucose absorption took place. Sodium and water uptake were reduced by glucagon administration; in all three groups there was a transient secretion of potassium into the intestinal lumen. These results, obtained after application of pharmacological doses, do not show clearly whether the demonstrated changes of resorption result directly from glucagon or from regulatory processes.

3816 EFFECTS OF BOVINE GROWTH HORMONE, HUMAN PLACENTAL LACTOGEN AND OVINE PROLACTIN ON INTESTINAL FLUID AND ION TRANSPORT IN THE RAT. *(E.)* Mainoya, J. R. (Dep. Zool., Univ. California, Berkeley). *Endocrinology* 96(5):1165-1170, 1975.

The influence of bovine growth hormone and human placental lactogen on intestinal absorption was compared with that of ovine prolactin. Normal and hypophysectomized adult male Sprague-Dawley rats were anesthesized and everted intestinal sacs were prepared from segments III and IV of the jejunum and from the entire colon. The sacs were filled with Ringer solution containing glucose and were placed in metabolic incubators; after incubation, mucosal transport was measured. One mg of bovine growth hormone, ovine prolactin, and/or purified human placental lactogen was injected s.c. 24 or 48 hr before surgery. Administration of each of these hormones resulted in increased fluid and electrolyte transport by the intestinal mucosa. Hypophysectomy produced a fall in fluid and ion absorption in the jejunum, but these changes were prevented by growth hormone administration. Bovine growth hormone and ovine prolactin produced essentially similar effects in intact rats: significant increases in fluid, sodium, and calcium transport in the duodenum; significant increases in fluid, sodium, and potassium transport in the jejunum; and significant increases in sodium, chloride, potassium, and calcium transport in the ileum. Growth hormone also significantly increased the transport of fluid, sodium, and chloride in the colon. Treatment of hypophysectomized rats with human placental lactogen enhanced fluid and ion transport in the jejunum but failed to restore normal potassium transport in the ileum and colon. The results suggest that growth hormone and human placental lactogen affect jejunal water and electrolyte transport in the same manner as does prolactin, possibly by influencing active ion transport.

3817 EFFECTS OF GASTROINTESTINAL HORMONES ON ADENYLATE CYCLASE ACTIVITY IN HUMAN JEJUNAL MUCOSA. *(E.)* Klaeveman, H. L.; Conlon, T. P.; Levy, A. G.; Gardner, J. D. (Natl. Inst. Arthritis, Metab. Dig. Dis., Bethesda, Md.). *Gastroenterology* 68(4): 667-675, 1975.

The effects of various polypeptide hormones on the adenylate cyclase activity in the human small intestinal mucosa were investigated to determine the mechanism by which such hormones alter the intestinal transport of salt and water. Peroral small intestinal biopsy specimens were obtained from 19 normal subjects of both sexes. Adenylate cyclase activity in the homogenized samples were determined by measuring the formation of ^{32}P-labeled cyclic AMP from ($\alpha^{32}P$)ATP in the presence of reaction mixtures containing the various hormones. Vasoactive intestinal polypeptide (VIP) and prostaglandin E_1 (PGE_1) activated adenylate cyclase at concentrations of 10^{-7} M, maximal stimulation occurring at concentrations of 10^{-4} M; the effects of these agents were additive. Neither glucagon nor secretin stimulated adenylate cyclase or altered the stimulation produced by VIP or PGE_1. Gastrin, cholecystokinin-octapeptide, pentagastrin, human calcitonin, and salmon calcitonin were also incapable of activating adenylate cyclase or altering the stimulating activity of PGE_1 or VIP. Several guanyl nucleotides, including guanosine monophosphate, guanosine diphosphate, guanosine triphosphate, and 5'-guanylyl imidodiphosphate, significantly increased basal adenylate cyclase activity but did not alter

the effects of PGE_1 or VIP. Several other nucleotide triphosphates (xanthosine triphosphate, inosine triphosphate, cytidine triposphate, uridine triphosphate) did not alter adenylate cyclase activity. These data suggest that VIP and PGE_1 act at functionally distinct sites and that the other polypeptide hormones tested do not interact with the receptors with which VIP and PGE_1 combine to increase adenylate cyclase activity.

3818 STUDIES ON THE METABOLISM OF DIMETHYLNITROSAMINE IN THE RAT. I. EFFECT OF DOSE, ROUTE OF ADMINISTRATION AND SEX. *(E.)* Phillips, J. C.; Lake, B. G.; Heading, C. E.; Gangolli, S. D.; Lloyd, A. G. (Br. Ind. Biol. Res. Assoc., Carshalton, Surrey, England). *Food Cosmet. Toxicol.* 13(2): 203-209, 1975.

Some of the factors affecting the metabolism of dimethylnitrosamine (DMN) in the rat were investigated. ^{14}C-DMN (0.2 ml/100 g) was administered i.p., s.c., i.v. and *via* oral intubation to mature and immature male and female specific-pathogen-free Wistar rats. The excretion of $^{14}CO_2$ was monitored. In other experiments, ^{14}C-DMN was injected s.c. or into the stomachs of pylorus-ligated rats and the absorption of ^{14}C-DMN from the stomach and small intestine was studied. Exhalation of unchanged ^{14}C-DMN accounted for less than 0.4% of the injected dose in the first experiment; the maximum rate of $^{14}CO_2$ production occurred between hr 1 and 4, less than 3% of the injected dose being exhaled between 6 and 24 hr. The maximum rate of $^{14}CO_2$ production was related to dose in both males and females, and the maximum rate of production was significantly lower in mature than immature animals. $^{14}CO_2$ production and the amount of ^{14}C excreted in the urine were similar for all p.o. administration but was significantly lower after p.o. administration. In the pylorus-ligated animals, the maximum rate of $^{14}CO_2$ production and the percentage $^{14}CO_2$ produced in 7 hr were only 45% of the control values. The disappearance of ^{14}C-DMN from the stomach was considerably smaller than from the small intestine, and absorption from the stomach, but not the small intestine, was significantly more rapid in starved than in fed rats. The percentage of ^{14}C remaining in the stomach did not appear to depend on the initial concentration of DMN. The kinetics of the disappearance of radioactivity from the small intestine varied with the intraluminal concentration of ^{14}C-DMN, following a monoexponential curve at very low concentrations and a biexponential curve at higher concentrations. The results suggest that the lower rate of DMN metabolism after oral administration is probably due to slower absorption of this compound from the stomach.

3819 ACTIVE AMINO-ACID AND SUGAR UPTAKE BY GALL BLADDER EPITHELIUM IN DOG, GUINEA-PIG AND MAN. *(E.)* Mirkovitch, V.; Sepulveda, F. V.; Menge, H.; Robinson, J. W. L. (Canton. Hosp., Lausanne, Switzerland). *Pflugers Arch.* 355(4):319-330, 1975.

The *in vitro* uptake of various sugars and amino acids by slices of canine gallbladder mucosa was studied. Samples of gallbladder and common bile duct from adult mongrel dogs, rabbits, guinea-pigs, and human beings were incubated in media containing ^{14}C-labeled substrates, and tissue uptake of radioactivity was determined by liquid scintillation counting. To determine the effect of bile retention on the transport capacity of the canine gallbladder mucosa, the common bile duct was ligated just proximal to the papilla vateri, and the gallbladder and bile duct were excised for examination 2 weeks later. The canine gallbladder was able to accumulate amino acids and sugars against considerable concentration gradients across the luminal membrane of the cell; the epithelium of the common bile duct also absorbed these compounds. The transport systems were sodium-dependent, saturable, and inhibited by ouabain and metabolic poisons (2,4-dinitrophenol and iodoacetic acid). Glycine was preferentially transported across the canine gallbladder from the mucosa to the serosa, but the net flux was small. Concentrative uptake also occurred in the guinea-pig and human gallbladders but appeared to be practically nonexistent in rabbit tissue. The gallbladder epithelium proved to be remarkably resistant to anoxia, and was able to concentrate amino acids and sugars, although to a significantly reduced extent, 2 weeks after obstruction of the common bile duct. The results indicate that the centraluminal membrane of the epithelial cell might be relatively impermeable to glycine and suggest a close similarity between the transport mechanism situated in the luminal membrane of the gallbladder and the proximal tubular cell of the kidney.

3820 MORPHOLOGICAL AND FUNCTIONAL BASIS OF DIGESTION AND ABSORPTION IN THE DIGESTIVE TRACT. *(Pol.)* Ziemlanski, S. (Inst. Physiol. Biochem., Warsaw, Poland). *Przegl. Lek.* 31(2):316-322, 1974.

3821 CONTROLLED AND UNCONTROLLED INFLUENCES ON DRUG AVAILABILITY. *(Ger.)* Speiser, P. (Inst. Pharm., Fed. Univ. Zurich, Switzerland). *Arzneim. Forsch.* 25(3):470-476, 1975.

3822 ABSORPTION AND SECRETION OF DRUGS BY THE MUCOSAL EPITHELIA OF THE GASTROINTESTINAL TRACT. *(Ger.)* Lauterbach, F. (Inst. Pharmacol. Toxicol., Ruhr Univ., Bochum, Germany). *Arzneim. Forsch.* 25(3):479-488, 1975.

3823 ABSORPTION, DISTRIBUTION AND EXCRETION OF [^{14}C]CTAB, A QUARTERNARY AMMONIUM SURFACTANT, IN THE RAT. *(E.)* Isomaa, B. (Inst. Biol., Abo Acad. Turku, Finland). *Food Cosmet. Toxicol.* 13(2): 231-237, 1975.

3824 ABSORPTION OF SELECTED FATTY ACIDS OF RAPESEED OIL IN THE ALIMENTARY TRACT. *(Pol.)* Ziemlanski, S.; Cieslakowa, D.; Okolska, G.; Kucharczyk, B. (Inst. Physiol. Biochem., Warzaw, Poland). *Przegl. Lek.* 31(2):322-327, 1974.

3825 pH AND BICARBONATE EXCRETION IN THE RAT PAROTID GLAND AS A FUNCTION OF SALIVARY RATE. *(E.)* Sommer, H. M.; Kaiser, D.; Drack, E. (Pediatr. Clin., Univ. Bern, Switzerland). *Pfluegers Arch.* 355 (4):353-360, 1975.

3826 ORGANIZATION OF THE BRUSH BORDER AND THE CONCEPT OF DIGESTION-ABSORPTION: RECENT FINDINGS ON ENTEROCYTE ULTRASTRUCTURE. *(Fr.)* Schmitz, J.; Jos, J.; Drillet, F.; Rey, F.; Rey, J. (Hosp. Sick Child., Paris, France). *Arch. Fr. Pediatr.* 31 (2/3):231-240, 1974.

3827 PARTICIPATION OF SUBCORTICAL NERVOUS STRUCTURES IN THE REGULATION OF INTESTINAL ABSORPTION. *(Rus.)* Faitelberg, R. O.; Vengrzhanovskii, P. N. (Dep. Hum. Anim. Physiol. I. I. Mechnikov State Univ. Odessa, USSR). *Fiziol. Zh. SSSR* 61(4):636-641, 1975.

3828 INTESTINAL IRON ABSORPTION IN ONE TO FOUR MONTH OLD INFANTS FED IRON-ENRICHED FORMULAS. *(Ger.)* Mahdi, S.; Heine, W. (Pediatr. Clin., Univ. Rostock, Germany). *Kinderaerztl. Prax.* 42(11): 505-508, 1974.

3829 INTESTINAL ABSORPTION OF CARBOHYDRATES AND TRIGLYCERIDES. *(Ger.)* Clodi, P. H. (Monk-Hospitaller Public Hosp., Linz, Austria). *Arzneim. Forsch.* 25(3):476-479, 1975.

3830 INFLUENCE OF HORMONES ON INTESTINAL TRANSFER OF WATER AND ELECTROLYTES. *(Ger.)* Ewe, K. (Med. Clin. I, Univ. Mainz, Germany). *Arzneim. Forsch.* 25(3):499-506, 1975.

3831 DUODENAL HORMONES AND THE POTENTIAL DIFFERENCE IN THE INTESTINE. *(Fr.)* Wingate, D. L.; Cooper, W. (London Hosp. Med. Coll., England). *Biol. Gastroenterol. (Paris)* 7(4):394, 1974.

3832 PENETRATION OF PROTEINS THROUGH INTESTINAL WALL OF PIGLETS BORN AT DIFFERENT TIMES DURING GESTATION. *(Ukr.)* Hrytsenko, N. M.; Kovalenko, V. F. (Poltava Res. Inst. Swine Breeding, USSR). *Ukr. Biokhim. Zh.* 46(1):79-83, 1974.

3833 THE INFLUENCE OF DRUGS ON THE ABSORPTIVE CAPACITY OF INTESTINAL MUCOSA. *(Ger.)* Caspary, W. F. (Univ. Med., Clin., Gottingen, Germany). *Arzneim. Forsch.* 25(3):489-498, 1975.

3834 AN ANOMALOUS EFFECT OF THE INTERMEDIATE PRODUCTS OF RIBOFLAVINE PHOTOLYSIS ON THE INTESTINAL ABSORPTION OF POORLY ABSORBED WATER-SOLUBLE DRUGS IN RATS. *(E.)* Ghazy, F. S.; Kimura, T.; Muranishi, S.; Sezaki, H. (Fac. Pharm. Sci., Kyoto Univ., Japan). *J. Pharm. Pharmacol.* 27(4):268-272, 1975.

3835 INTESTINAL FUNCTION IN CHRONIC ALCOHOLISM. *(E.)* Mezey, E. (Baltimore City Hosp., Md.). *Ann. NY Acad. Sci.* 252:215-227, 1975.

3836 INHIBITION BY CAFFEINE OF ETHANOL ABSORPTION IN RATS. *(It.)* Siegers, C. P.; Strubelt, O.; Back, G. (Med. Acad., Lubecka, Germany). *Arch. Sci. Med. (Torino)* 131(3):173-180, 1974.

3837 BILE ACIDS AND ACTIVE TRANSPORT OF SUGARS AND AMINO ACIDS. *(Ger.)* Caspary, W. F. (Med. Clin., Univ. Gottingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:412-415, 1974.

3838 RADIOISOTOPE METHOD IN THE STUDY OF THE ABSORBING CAPACITY OF SEPARATE SEGMENTS OF THE SMALL INTESTINE. *(Rus.)* Kosharko, K. A.; Volkov, D. M.; Nalivkina, A. I.; Telnov, Y. A. (Inst. Roentgenol. Med. Radiol., Borisov, USSR). *Med. Radiol. (Mosk.)* 19(8):17-20, 1974.

3839 EFFECTS OF HUMAN SYNTHETIC GASTRIN I ON WATER, ELECTROLYTE, AND GLUCOSE ABSORPTION IN THE HUMAN SMALL INTESTINE. *(Fr.)* Modigliani, R.; Mary, J. Y.; de Peyer, R.; Bernier, J. J. (St. Lazare Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4):309-310, 1974.

3840 ABSORPTION AND SECRETION BY THE COLON. *(E.)* Cummings, J. H. (Cent. Middlesex Hosp., London, England). *Gut* 16(4):323-329, 1975.

3841 THE EFFECT OF A DEPHENOLIC LAXATIVE (BISACODYL) ON WATER AND ELECTROLYTE TRANSPORT IN THE HUMAN COLON. *(Ger.)* Ewe, K.; Holker, B. (1st Med. Polyclin., Univ. Mainz, Germany). *Klin. Wochenschr.* 52(17):827-833, 1974.

3842 ION TRANSPORT BY AMPHIBIAN ANTRUM IN VITRO. I. GENERAL CHARACTERISTICS. *(E.)* Flemstrom, G.; Sachs, G. (Lab. Memb. Biol., Univ. Alabama, Birmingham). *Am. J. Physiol.* 228(4):1188-1198, 1975.

3843 QUANTITATION OF CONDUCTANCE: PATHWAYS IN ANTRAL GASTRIC MUCOSA. *(E.)* Spenney, J. G.; Flemstrom, G.; Shoemaker, R. L.; Sachs, G. (VA Hosp., Birmingham, Alabama). *J. Gen. Physiol.* 65 (5):645-662, 1975.

3844 THE EFFECTS OF ACUTE ALTERATIONS IN HEMODYNAMICS, OXYGEN AVAILABILITY AND ACID BASE BALANCE ON THE PERMEABILITY OF THE GASTRIC MUCOSA. *(E.)* Ritchie, Jr., W. P. (Dep. Surg., Univ. Charlottesville, Va.). 7 pp., 1975. [available through National Technical Information Services, Washington, D.C. Document No. AD-A008 093/7WJ]

3845 CALCIUM ABSORPTION AS A FUNCTION OF CALCIUM INTAKE. *(E.)* Heaney, R. P.; Saville, P. D.; Recker, R. R. (Creighton Univ. Sch. Med., Omaha, Neb.). *J. Lab. Clin. Med.* 85(6):881-890, 1975.

3846 TIN — RESORPTION, PERORAL TOXICITY AND MAXIMUM ADMISSABLE CONCENTRATION IN FOODS. *(E.)* Knorr, D. (Inst. Food Technol., Sch. Soil Cult., Vienna, Austria). *Lebensm. Wiss. Technol.* 8(2):51-56, 1975.

3847 BACKGROUND CONSIDERATIONS OF INTESTINAL CALCIUM ABSORPTION AND ITS RELATION TO VITAMIN D. *(E.)* Norman, A. W. (no affil.). In: *Vitamins and Hormones. Advances in Research and Applications.* edited by Harris, R. S. (New York: Academic Press, 1974).

3848 SULPHATE TRANSPORT BY RAT ILEUM. EFFECT OF MOLYBDATE AND OTHER ANIONS. *(E.)* Cardin, C. J.; Mason, J. (Dep. Biochem., Trinity Coll., Dublin, Ireland). *Biochim. Biophys. Acta* 394(1):46-54, 1975.

3849 INHIBITION OF PYRUVATE TRANSPORT BY FATTY ACIDS IN ISOLATED CELLS FROM RAT SMALL INTESTINE. *(E.)* Lamers, J. M. J.; Hulsmann, W. C. (Fac. Med., Erasmus Univ., Rotterdam, Netherlands). *Biochim. Biophys. Acta* 394(1):31-45, 1975.

3850 NATURE AND IMPORTANCE OF ENDOGENOUS FATTY ACIDS DURING INTESTINAL ABSORPTION OF FATS. *(E.)* Clement, J. (Natl. Cent. Sci. Res., Dijon, France). *World Rev. Nutr. Diet.* 21:281-307, 1975.

3851 EFFECT OF FREE GOSSYPOL ON TISSUE UPTAKE OF IRON BY EVERTED SEGMENTS OF RAT DUODENUM. *(E.)* Herman, D. L.; Smith, F. H. (Dep. Anim. Sci., North Carolina State Univ., Raleigh). *J. Agric. Food Chem.* 23(3):548-551, 1975.

3852 BILIARY EXCRETION AND ENTEROHEPATIC CIRCULATION OF PREGNANOLONE IN THE RAT. *(E.)* Long, R. J.; Soyka, L. F. (Univ. Illinois Coll. Med., Chicago). *Biochem. Pharmacol.* 24(10):1067-1072, 1975.

3853 VITAMIN D: 3-DEOXY-1α-HYDROXYVITAMIN D_3, BIOLOGICALLY ACTIVE ANALOG OF 1α,25-DIHYDROXYVITAMIN D_3. *(E.)* Norman, A. W.; Mitra, M. N.; Okamura, W. H.; Wing, R. M. (Dep. Biochem., Univ. California, Riverside). *Science* 188(4192):1013-1015, 1975.

3854 EFFECT OF NAFENOPIN (SU-13,437) ON LIVER FUNCTION. HEPATIC UPTAKE AND BILIARY EXCRETION OF OUABAIN IN THE RAT. *(E.)* Meijer, D. K. F.; Bognacki, J.; Levine, W. G. (Albert Einstein Coll. Med., Yeshiva Univ., Bronx, N.Y.). *Drug Metab. Dispos.* 3(3):220-225, 1975.

3855 EFFECT OF CALCIUM AND PHOSPHORUS ON FLUORIDE METABOLISM IN MAN. *(E.)* Spencer, H.; Osis, D.; Kramer, L.; Wiatrowski, E.; Norris, C. (VA Hosp., Hines, Ill.). *J. Nutr.* 105(6):733-740, 1975.

3856 SODIUM CHLORIDE TRANSPORT BY RABBIT GALLBLADDER. DIRECT EVIDENCE FOR A COUPLED NaCl INFLUX PROCESS. *(E.)* Frizzell, R. A.; Dugas, M. C.; Schultz, S. G. (Univ. Pittsburgh, Pa.). *J. Gen. Physiol.* 65(6):769-795, 1975.

3857 BILIARY EXCRETION OF [^{3}H]ALDOSTERONE AND ITS SEX DEPENDENCE IN ADRENALECTOMIZED RATS. *(E.)* Morris, D. J.; Silverman, J. A. (Miriam Hosp., Providence, R.I.). *Endocrinology* 96(6):1386-1391, 1975.

3858 CONCENTRATION DEPENDENCE OF ETHANOL. EFFECT ON INTESTINAL ABSORPTION OF THEOPHYLLINE IN RATS. *(E.)* Harrigan, D. J.; Levy, G. (Sch. Pharm., State Univ. New York, Buffalo). *J. Pharm. Sci.* 64(5): 897-898, 1975.

3859 BILE ACIDS: THEIR ABSORPTION FROM THE GASTROINTESTINAL TRACT AND ROLE DURING FAT ABSORPTION. *(E.)* Dietschy, J. M. (Univ. Texas Southwest. Med. Sch., Dallas). *Verh. Dtsch. Ges. Inn. Med.* 80:399-407, 1974.

3860 EFFECT OF FASTING AND ANTINEOPLASTIC AGENTS ON THE INTESTINAL ABSORPTION OF DRUGS IN THE RATS. *(E.)* Komuro, T.; Kitazawa, S.; Sezaki, H. (Hosp. Pharm., Kyoto Univ., Japan). *Chem. Pharm. Bull.* 23(2): 400-408, 1975.

See also, 3791, 3800, 3877, 4138, 4152, 4153, 5067, 5091.

MOTILITY

3861 EFFECT OF ISCHEMIC ANOXIA ON MYOELECTRICAL AND MECHANICAL ACTIVITY OF THE TOTALLY ISOLATED CANINE STOMACH. *(E.)* Kowalewski, K.; Zajec, S.; Kolodej, A. (Surg.-Med. Res. Inst., Univ. Alberta, Edmonton, Canada). *Surg. Gynecol. Obstet.* 140(2):187-192, 1975.

Electrical and mechanical activities were recorded from the totally isolated canine stomach perfused with homologous blood of a living oxygenator-supported dog. Stagnant ischemic anoxia was induced by closing inflow and outflow blood from the isolated organ for various periods up to 4 hr. Recording of electrical and mechanical activity was performed prior to, during, and after the anoxic periods. After a given period of time, blood was recirculated for 10 min and pentagastrin (5 μg) was injected into the gastric artery. Ischemia induced first low frequency electrical control activity, and it altered the direction of conduction and phase lag of control activity. Contractions, if present prior to ischemia, were abolished after 10-20 min of circulatory arrest. No signals were recorded from electrically active areas of the stomach after 30-50 min of ischemia. After up to 3 hr of anoxia, stomachs showed recordable electrical activity when perfused for 10 min with homologous blood and essentially normal electrical control activity and mechanical activity 10 min after pentagastrin injection. After 3.5-4 hr of anoxia, recirculation of blood and pentagastrin had no effect on electrical and mechanical activity of the gastric musculature. That *ex vivo* canine stomachs can tolerate 3 hr of stagnant ischemia suggests that metabolic function in the smooth muscle cells, responsible for the periodic oscillations in potential, is not irreversibly damaged after this period of ischemia. The response to pentagastrin indicates that functional integrity of the intramural plexus was not destroyed by 3 hr of anoxia and that the nerve plexus layers were still able to synthesize and release acetylcholine.

3862 GASTROESOPHAGEAL DYNAMICS DURING IMMERSION IN WATER TO THE NECK. *(E.)* Johnson, L. F.; Lin, Y. C.; Hong, S. K. (Tripler Army Med. Cent., Honolulu, Hawaii). *J. Appl. Physiol.* 38(3):449-454, 1975.

The gastroesophageal pressure gradient, distal esophageal sphincter pressure, and the gastroesophageal pH gradient were studied in man during immersion to the neck in water, and standing in air with and without abdominal compression. An esophageal pressure probe and pH electrode were passed into the esophagus of five subjects while standing in air. The catheter assembly was advanced in 1 cm increments until all gauges had traversed the distal esophageal sphincter and entered the stomach. The instrument was then withdrawn slowly until the proximal pressure gauge had entered the hypopharynx. During the measurements the subject was encouraged not to swallow. Identical procedures were repeated while the subject was standing in water up to the neck, and was standing in air with a gastric pressure of 20 mm Hg created by a thigh blood pressure cuff around the abdomen. While standing in air, gastric pressure averaged 4.6 mm Hg while the esophageal sphincter pressure was 19.4 mm Hg. The gastroesophageal pressure gradient was 58 mm Hg. During immersion in water, gastric pressure increased to nearly 20 mm Hg and esophageal sphincter to about 32 mm Hg. Esophageal pressure was about 5 mm Hg higher than the corresponding in air measurements, and the point of lowest pressure shifted proximally. While standing in air with abdominal compression, gastric and sphincter pressures were similar to those in immersed subjects, but esophageal pressure was midway between values in immersed subjects and in air subjects. The pH abruptly changed from nearly neutral in the esophagus to strongly acidic as the catheter traversed the sphincter. The location where the pH had been 6 shifted 4 cm proximally during immersion. The esophagus shortened in length between the cricopharyngeus and the upper limit of the sphincter by 11% during immersion and 9.7% during abdominal compression in air. These two values did not differ significantly from each other. Although no evidence of reflux was found, it is suggested that immersion predisposes to gastric reflux in those with low sphincter tone.

3863 EFFECTS OF CAERULEIN ON THE ISOLATED HUMAN ILEUM. *(E.)* del Tacca, M.; Soldani, G.; Crema, A. (Dep. Pharmacol. Univ. Pisa, Italy). *Rend. Gastroenterol.* 6(3):165-167, 1974.

The effects of cerulein on the human intestine *in vitro* was studied using longitudinal strips removed during surgical operations from patients of both sexes. Before beginning the experiments, the preparation was left standing for about 120 min, by which time spontaneous activity had developed; at this time the effectiveness of transmural stimulation was determined with trains of pulses of 1 μsec duration at a frequency of 10/sec lasting for 30 sec. Cerulein produced a pronounced and sustained contraction of the longitudinal muscle, which relaxed after washing. Tachyphylaxis readily occurred. The threshold concentration differed from one organ to another, but in all the experiments cerulein was used at 1×10^{-8} g/ml. In the presence of atropine (5×10^{-7} g/ml), the contraction in response to transmural stimulation was abolished and replaced by a biphasic response. Bradykinin (5×10^{-8} g/ml), used as a reference drug for its direct action on the muscle, was unaffected. Tetrodotoxin (1×10^{-6} g/ml) added in the presence of atropine caused a further but not significant reduction of response to cerulein. The response to bradykinin was not affected by atropine and tetrodotoxin. In the presence of atropine plus tetrodotoxin, the response to cerulein was resumed at higher doses of polypeptide (1×10^{-6} g/ml). Hexamethonium (1×10^{-4} g/ml) reduced the response to cerulein by 25%, while bradykinin was not affected. The results indicate that the effects of cerulein on isolated human ileum are mediated mainly through cholinergic structures because atropine reduces its response. The stimulant effect of cerulein, however, appears to be at least partially independent of the cholinergic nervous system, because neither atropine nor tetrodotoxin completely abolished the contractions. The response to cerulein was only partially affected by hexamethonium, indicating that cerulein stimulates mainly at postganglionic sites. It is suggested that response remaining after atropine and tetrodotoxin is either directly on the smooth muscle cells or mediated through the release of a mediator at the level of the nerve endings.

3864 COMPARATIVE STUDIES ON THE EFFECTS OF BRADYKININ AND VAGAL STIMULATION ON MOTILITY

IN THE STOMACH AND COLON. *(E.)* Fasth, S.; Hulten, L.; Jahnberg, T.; Martinson, J. (Sahlgrenska Hosp., Univ. Goteburg, Sweden). *Acta Physiol. Scand.* 93(1): 77-84, 1975.

The effect of bradykinin on gastric and colonic motility was compared with the effects of vagal nerve stimulation in cats. Eighteen cats were anaesthesized and fitted with volume recording devices which allowed intragastric and intraluminal pressures to be kept constant. Intraaortic bradykinin (10 µg) caused a profound and prolonged gastric relaxation along with a marked and prolonged colonic contraction. The gastric relaxation closely mimicked the atropine resistant relaxation elicited by vagal stimulation. Bradykinin did not reproduce the vagal motility effects on colon smooth muscle but mimicked the atropine resistant expulsive contraction elicited by activiation of the pelvic nerves. The effects were not blocked by anti-adrenergic drugs. The results suggest that the gastric relaxation elicited by intraaortic bradykinin is not caused by an adrenergic mechanism or by a direct ganglion stimulating effect but is due to a direct effect on the gastric smooth muscles; further investigations are suggested to determine whether a kinin-like substance is involved.

3865 ELECTROMYOGRAPHY OF THE SPHINCTER OF ODDI: TECHNIC AND EXPERIMENTAL RESULTS IN THE RABBIT: EFFECT OF CERTAIN DRUGS. *(E.)* Sarles, J. C.; Midejean, A.; Devaux, M. A. (Dig. Pathol. Res. Unit, INSERM, Marseille, France). *Am. J. Gastroenterol.* 63(3):221-231, 1975.

Electromyographic tracings of the rabbit sphincter of Oddi *in vivo* (50 rabbits) and *in vitro* (20 rabbits) were described, in addition to the effect of alcohol and various pharmacodynamic agents on the choledochoduodenal junction. Electromyographic activity was recorded by means of bipolar suction electrodes placed on the sphincter of Oddi and the adjacent duodenum. The bile duct was catheterized so that the pressure could be recorded. The sphincteric traces showed basal activity consisting of bursts of 1-8 spike potentials every 3-5 sec accompanied by a transient rise in biliary pressure. The patterns were different from those recorded for the duodenum in which the biliary pressure was not affected. The frequency and amplitude of the spike potentials increased with temperature and the rise in biliary perfusion pressure. Acetylcholine (1.8 mg/kg, i.v.), prostigmin (0.05 mg/kg, i.v.), and morphine hydrochloride (0.4 mg/kg, i.v.) increased the frequency and amplitude of the spike potentials and shortened the resting intervals. Inhaled amyl nitrite and i.v. hexamethonium (2 mg/kg/hr) reduced electrical activity. Atropine (0.2 mg/kg, i.v.) had a very slight depressant effect. Alcohol (1 g/kg) administered as an i.v. drip resulted in a substantial increase in the electrical activity of the sphincter and in biliary pressure. The effect was reduced by vagotomy and suppressed by hexamethonium (1 mg/kg/hr, i.v.) given 30 min before alcohol. The results suggest that the sphincter of Oddi is the site of basal contractile activity, consisting of rhythmic muscular contractions, which prevent the discharge of bile into the duodenum and temporarily raise the pressure in the lower end of the common bile duct.

3866 MOTILITY CHANGES IN THE ANTRUM AFTER PROXIMAL GASTRIC VAGOTOMY. *(E.)* Kelly, M.; Kennedy, T. (R. Victoria Hosp., Belfast, Ireland). *Br. J. Surg.* 62(3):215-220, 1975.

The motility of the antrum in the normal animal was studied before and after truncal, selective and proximal gastric vagotomy. The resting and postprandial motor activity in the gastric antrum was observed in 6 dogs using a miniaturized mercury column strain gauge. There was a gradual increase in the amplitude of contraction during the first 2 hr after eating, which was maintained for 3 hr, and then declined. For the first 45 min, terminal antral contraction occurred, partially retaining and triturating the gastric contents. After 45 min, the waves became sequential, symmetrical, increased in vigor, and actively pumped food into the duodenum. Truncal and selective vagotomy reduced the work capability to 20% of normal 1 month after operation; the waves were disorganized. Proximal gastric vagotomy abolished the braking mechanism and removed the initial inhibitory stimuli to antral motility. Within 1 month, the antrum regained 58% of its normal work capability and the contractions were well organized. The results indicate that the avoidance of a drainage operation in combination with proximal gastric vagotomy maintains an active and effective pyloric pump which recovers rapidly after operation.

3867 NEURAL ORGANIZATION OF ESOPHAGEAL PERISTALSIS: ROLE OF VAGUS NERVE. *(E.)* Mukhopadhyay, A. K.; Weisbrodt, N. W. (Univ. Texas Med. Sch., Houston). *Gastroenterology* 68(3): 444-447, 1975.

A study was undertaken to determine the velocity of peristalsis in the smooth muscle area of the opossum esophagus before and after atropine administration and to evaluate the role of the vagus nerves in controlling the propagation of esophageal peristalsis. Tracheostomies were performed on animals of both sexes and the intraluminal pressures in the esophagus were measured with a pressure transducer recorder system. The velocity of peristalsis in the lower third of the esophagus was measured before and after atropine (100 µg/kg, i.p.) following the induction of swallowing by pharyngeal stimulation. In another group of animals, the vagus nerves were sectioned behind and between the carotid artery and jugular vein, and the esophageal contractions produced by electrical stimulation were measured after i.p. atropine (100 µg/kg) administration. The velocity of peristalsis in the lower third of the esophagus progressively decreased from 3.25 cm/sec to 1.83 cm/sec at the lower esophageal sphincter. After atropine administration, the velocity decreased from 3.1 cm/sec to 1.74 cm/sec at the lower esophageal sphincter; these changes were not significant. Electrical stimulation of the distal cut end of the vagus nerve induced peristaltic contractions, the velocities of

peristalsis after electrical stimulation decreasing from 3.24 cm/sec to 1.84 cm/sec at the lower esophageal sphincter. The results indicate that the velocity of peristalsis in the smooth muscle area of the opossum esophagus has a caudally decreasing gradient. Bilateral cervical vagotomy and stimulation of the distal cut end initiated peristaltic contraction, indicating that the propagative nature of peristalsis in the smooth muscle area of the opossum esophagus has a caudally decreasing gradient. Bilateral cervical vagotomy and stimulation of the distal cut end initiated peristaltic contraction, indicating that the propagative nature of peristalsis in the smooth muscle segment of the body of the esophagus is controlled by a local mechanism within the esophagus rather than within the central nervous system.

3868. EFFECT OF VAGOTOMY ON ELECTRICAL ACTIVITY OF THE SMALL INTESTINE OF THE DOG. *(E.)* Weisbrodt, N. W.; Copeland, E. M.; Moore, E. P.; Kearley, R. W.; Johnson, L. R. (Univ. Texas Med. Sch., Houston). *Am. J. Physiol.* 228(2):650-654, 1975.

The effect of bilateral thoracic vagotomy on the myoelectric activity of the small intestine was studied in conscious male mongrel dogs. The animals were implanted with electrodes spaced 25 cm apart along the serosal surface of the small intestine, and a cannula was placed in the most dependent portion of the stomach. Recordings were made with dogs in fasted and fed states. Two distinct patterns of myoelectric activity were recorded: one typical of the fasted state (the interdigestive myoelectric complex) and one typical of the fed state. After completion of the control recording periods, a truncal vagotomy was performed on each animal. Completeness of the vagotomy was confirmed by lack of a gastric secretory response to insulin. Gastric stasis occurred after vagotomy, so animal stomachs were emptied *via* the gastric cannula to obtain a fasted condition. Vagotomy had little or no effect on the fasted pattern of myoelectric activity, but the fed pattern was significantly altered in 2 animals. This alteration might have been due to the effect of vagotomy on gastrin release. It is concluded that nervous pathways within the vagus may exert some influence on the intestinal myoelectric activity, but that other neural-humoral pathways are probably also involved.

3869 EFFECT OF PROSTAGLANDINS E_1, E_2 AND $F_{2\alpha}$, AND PENTAGASTRIN ON THE GALLBLADDER PRESSURE IN DOGS. *(E.)* Nakano, J.; McCloy, Jr. R. E.; Gin, A. C.; Nakano, S. K. (Univ. Oklahoma Coll. Med., Oklahoma City). *Eur. J. Pharmacol.* 30(1):107-112, 1975.

The effects of pentagastrin and prostaglandins E_1 (PGE_1), E_2, (PGE_2), and $F_{2\alpha}$ ($PGE_{2\alpha}$) on gallbladder pressure were studied in anesthetized dogs. The gallbladders of 34 dogs were exposed through an upper midline laparotomy. The various drugs were injected into a femoral vein and the effects on gallbladder and systemic arterial pressures monitored continuously. In some animals, the cystic duct was clamped. Both PGE_1 and PGE_2 (4 µg/kg) lowered gallbladder pressure when the initial pressure was higher than 5 mm Hg, but caused no significant change when the initial pressure was lower than 3 mm Hg; both significantly decreased the systemic arterial pressure. In contrast, $PGF_{2\alpha}$ (4 µg/kg) increased systemic arterial and gallbladder pressures regardless of the initial pressure. When the cystic duct was clamped, $PGF_{2\alpha}$ markedly increased gallbladder pressure, while both PGE_1 and PGE_2 increased it only slightly. Furthermore, $PGF_{2\alpha}$ appeared to constrict the sphincter of Oddi, while PGE_1 and PGE_2 relaxed it. Pentagastrin (200 µg/kg) initially decreased then slightly increased systemic arterial pressure and produced a significant monophasic decrease in gallbladder pressure regardless of the initial pressure. Pretreatment with indomethacin did not affect or slightly potentiated the cholecystokinetic effect of the prostaglandins, but abolished that of pentagastrin. The results suggest that gallbladder smooth muscle is markedly stimulated by $PGF_{2\alpha}$ and stimulated to a lesser degree by $PGE_1$and PGE_2. They also suggest that prostaglandins are possible mediators for the cholecystokinetic action of the gastrointestinal hormones.

3870 EFFECTS OF ACETYLCHOLINE RELEASING DRUGS ON ELECTRICAL ACTIVITIES OBTAINED FROM AUERBACH'S PLEXUS IN THE GUINEA PIG ILEUM. *(E.)* Sato, T.; Takayanagi, I.; Takagi, K. (Fac. Pharm. Sci., Univ. Tokyo, Japan). *Jpn. J. Pharmacol.* 24(3): 447-451, 1974.

The effects of 5-hydroxytryptamine, sodium picrate, cerulein, and pentagastrin on the electical activities in Auerbach's plexus in the guinea pig ileum were studied. The ileal longitudinal muscle with Auerbach's plexus was removed from male animals and suspended in an organ bath filled with Locke-Ringer's solution. The drugs and various antagonists (atropine sulfate, hexamethonium bromide, nicotine bitartrate, and morphine hydrochloride) were injected into the bath and the responses of the tissue recorded using an isotonic lever. At 3×10^{-7} to 3×10^{-6} g/ml, 5-hydroxytryptamine increased the spike frequency in Auerbach's plexus; this was unaffected by pretreatment with atropine, hexamethonium, or nicotine. Sodium picrate (10^{-4} g/ml), cerulein (3×10^{-9} g/ml), and pentagastrin (5×10^{-7} g/ml) also increased the spike frequency, their effects similarly being little affected by atropine or hexamethonium. Tachyphylaxis was observed following repeated administration of pentagastrin at 10^{-6} g/ml, and large spikes were often simultaneously recorded with 5-hydroxytryptamine, cerulein, pentagastrin, and sodium picrate. The large spikes were inhibited by atropine and Mn^{++}, but not by tetrodotoxin. Morphine reduced the frequency of the single spikes elicited by 5-hydroxytrypamine as well as the 5-hydroxytryptamine-induced contraction of the isolated longitudinal muscle strip; morphine had a similar ef-

fect on the responses elicited by cerulein. The results suggest that the sites of action of these agonists are in Auerbach's plexus and that the agonists modify to a minimum extent the electrical activities of Auerbach's plexus *via* muscarinic and nicotinic receptors. Furthermore, these agonists probably accelerate the release of acetylcholine and contract the ileum.

3871 MECHANICAL RESPONSES TO CATECHOLAMINES IN ISOLATED STRIPS OF THE GUINEA-PIG STOMACH MUSCLE. *(E.)* Yamaguchi, T.; Tomita, T. (Fac. Med., Kyushu Univ., Fukuoka, Japan). *Jpn. J. Pharmacol.* 24(6):911-922, 1974.

The effects of various catecholamines on the mechanical responses caused by electrical stimulation of excitatory and inhibitory nerves in the guinea pig stomach were studied. Strips of circular and longitudinal muscle from animals of both sexes were suspended in organ baths containing Krebs solution. Epinephrine hydrochloride, tetrodotoxin citrate, norepinephrine hydrochloride, isoproterenol hydrochloride, phentolamine mesylate, propranolol hydrochloride, and hyoscine butylbromide were injected into the organ baths, the blockers being applied 10-15 min prior to catecholamine application. The preparations were stimulated between two Ag-AgCl electrodes placed at opposite ends of the bath. In the circular muscles, the relaxation and biphasic response elicited by epinephrine and norepinephrine were partially suppressed by phentolamine and propranolol; the catecholamine-induced relaxation in the longitudinal muscles was inhibited by propranolol. α-Relaxation was more dominant in the circular muscles, while β-relaxation was more dominant in the longitudinal muscles. The contractions in both muscle types were mediated *via* α-effects, but the circular muscle was more sensitive to the α-effect while the longitudinal muscle was more sensitive to the β-effect. In the presence of propranolol, isoproterenol caused a monophasic contraction or a biphasic response in both circular and longitudinal muscle. The inhibition of the electrically evoked contractions by epinephrine and norepinephrine was abolished by phentolamine in both muscle types, but the electrically evoked relaxations were little affected. The data suggest that the suppressive action of the catecholamines is probably due to the prevention of acetylcholine release by activation of the α-receptors in the cholinergic nerves. The suppression of evoked contractions by isoproterenol was probably due to a direct action on the muscle rather than an effect on nervous elements.

3872 EFFECTS OF GASTROINTESTINAL HORMONES ON VASCULAR SMOOTH MUSCLE. *(E.)* Fara, J. W. (Health Sci. Cent., State Univ. New York, Stony Brook). *Am. J. Dig. Dis.* 20(4):346-353, 1975.

The effects of secretin and cholecystokinin (CCK) on the mechanical activity of the rat portal vein were investigated and compared with those of isoproterenol, theophylline, and cyclic and dibutyryl cyclic AMP (cAMP). Thirty-three portal veins from male and female rats were mounted in Krebs-solution baths containing the various drugs being tested; some of the solutions contained 4-5 times the normal potassium ion concentration. Boots secretin (0.03-0.60 U/ml), pure natural secretin (0.18-0.60 U/ml), and CCK (0.12-0.60 U/ml) elicited dose-dependent decreases in the amplitude and simultaneous increases in the frequency of the spontaneous venous contractions. These effects were mimicked by isoproterenol, theophylline, and cAMP. All agents tested were also effective in reducing potassium- or norepinephrine-induced tension. In addition, subthreshold doses of CCK and secretin elicited responses when given in combination, and the effects of the hormones at all dose levels were potentiated by theophylline. The mechanical responses to secretin, CCK, and isoproterenol were unaffected by atropine and phenoxybenzamine, and only the response to isoproterenol was blocked by propranolol. The data indicate that secretin and CCK have a direct inhibitory effect on rat portal vein activity and that this effect is not mediated through α- or β-adrenergic or cholinergic receptors. They also suggest that CCK, secretin, and isoproterenol exert their action on the portal vein through a cAMP mechanism. At least a part of the mechanism of hormone-induced mesenteric vasodilatation may involve a direct relaxing effect of the hormones on the vascular smooth muscle.

3873 A RAPID PULL-THROUGH TECHNIQUE FOR MEASURING LOWER ESOPHAGEAL SPHINCTER PRESSURE. *(E.)* Dodds, W. J.; Miller, W. N.; Lydon, S. B.; Arndorfer, R. C. (Med. Coll. Wisconsin, Milwaukee). *Gastroenterology* 68(3):437-443, 1975.

Measurement of lower esophageal sphincter pressure by rapid pull-through technique was compared with that by conventional station pull-through technique. Twenty-four studies were performed on 12 normal volunteers using a 5-lumen catheter assembly. Sphincter pressure was measured by the conventional method, in which the catheter radial recording tips were withdrawn through the sphincter in 0.5 cm increments with a 10-sec pause at each station, and the rapid method, in which the sensors were withdrawn manually at 0.5 -1.0 cm/sec during a 10-15 sec interval of suspended respiration. Two complete sets of studies were done 2-4 wk apart. First-study results from each subject were scored independently by different observers on 2 separate occasions, while those of the second study were scored once only by each observer. Intraobserver and interobserver scoring errors were calculated, and a variance analysis was done. In the 1st study, differences in intraobserver error were small, but were significantly less in the rapid technique (0.3) than the standard (1.3). Interobserver scoring error was greater for results from the standard technique than the rapid. Scoring errors were not sufficient to obscure pressure differences between subjects. Variance between intrasubject pull-throughs was significantly greater for standard (10.0-6.6 mm Hg) than for rapid (7.3-4.3 mm Hg) techniques. Data from the first and second studies showed pressures calculated from the rapid method to be slightly greater than those from the stan-

dard method (24.3 and 21.1 mm Hg, resp)—a significant difference. Calculation from either method correlated significantly between studies. It is concluded that intraobserver and interobserver scoring errors are less for recorded and calculated pressure obtained by the rapid method; the sphincter-gastric pressure gradient is slightly greater when calculated from the rapid method; and calculated pressure is reproducible for both methods.

3874 PLASMA CATECHOLAMINES AND POSTOPERATIVE GASTRIC EMPTYING AND SMALL INTESTINAL PROPULSION IN THE RAT. *(E.)* Dubois, A.; Henry, D. P.; Kopin, I. J. (Natl. Cancer Inst., Bethesda, Md.). *Gastroenterology* 68(3):466-469, 1975.

The role of the adrenal medullary discharge of catecholamines in the inhibition of gastric emptying and small intestinal propulsion after laparotomy was examined using male Sprague-Dawley rats. Fasted demedullated and sham-demedullated rats were anesthetized and the small intestine and colon exteriorized, wrapped in gauze, and replaced after 30 min. Twelve hr later, 2 ml of a ^{51}Cr-labeled liquid test meal were administered *via* gastric tube and the rates of gastric emptying and propulsion of the meal into the distal small intestine were determined. The concentration of plasma catecholamines was determined by an enzymatic assay. Fourteen days after adrenal demedullation, the plasma catecholamine levels were significantly lower than in the sham-demedullated animals. Twelve hr after exteriorization of the small intestine, there was a significant increase in the plasma catecholamine levels of the sham-demedullated rats but not the demedullated rats. Gastric emptying before and after exteriorization of the intestine occurred at the same rate in the demedullated and sham-operated rats. Similarly, the 2 groups did not differ in the rate of small intestinal propulsion after a test meal; in both groups, the rate of transfer of the intestinal contents to the distal portion of the intestine was significantly retarded 12 hr after intestinal exteriorization. The results indicate that *in vivo*, circulating catecholamines have only a minor influence on gastric emptying and intestinal propulsion of a test meal.

3875 MODIFICATION OF 5-HT ACTION BY MAGNESIUM IONS ON THE GUINEA PIG ILEUM. *(E.)* Singh, G. (Lady Hardinge Med. Coll., New Delhi, India). *Jpn. J. Pharacol.* 24(4):639-640, 1974.

The effects of different perfusion media on the responses of the guinea pig ileum to 5-hydroxytryptamine (5-HT) were studied. Ileal segments from mongrel guinea pigs were perfused with Tyrode's solution or mammalian Ringer's solution. Of 10 pieces of intestine from different animals, seven showed tachyphylaxis to 5-hydroxytryptamine when perfused with Tyrode's solution; the height of the contractions were increased substantially by placing the segments in the Ringer's medium. Subsequently, the action of graded doses of 5-hydroxytryptamine was studied in ileal segments perfused with Ringer's solution to which $MgCl_2$ had been added. $MgCl_2$ ($1\text{-}2 \times 10^{-4}$ g/ml) reduced or completely blocked the response to all doses of 5-hydroxytryptamine; it also reduced the response of the ileal segments to histamine. 5-Hydroxytryptamine failed to contract segments of depolarized guinea pig ileum perfused with K_2SO_4-Ringer's solution to which $CaCl_2$ had been added. The results suggest that 5-hydroxytryptamine may make calcium available for the contractile machinery of the guinea pig ileum by a process of depolarization. Antagonism between 5-hydroxytryptamine and magnesium may be secondary to an antagonism between calcium and magnesium.

3876 LOCAL SPECIALIZATION AT ILEOCECAL JUNCTION OF THE CAT AND OPOSSUM. *(E.)* Conklin, J. L.; Christensen, J. (Univ. Hosp., Univ. Iowa, Iowa City). *Am. J. Physiol.* 228(4):1075-1081, 1975.

Characteristics which distinguish the ileocecal junction of the opossum from that of the cat were studied in 10 mature animals of each species. Serial 2-mm-wide transverse strips of muscularis propria were prepared from samples of ileum and colon taken from the region 1.5 cm above and about 1.5 cm below the ileocecal mucosal junction. These strips were separately stretched to determine the relation between length and tension and were separately subjected to electrical field stimulation. Anatomically, there was a thickening of the circular muscle layer in the ileal wall above the ileocolic mucosal junction in the cats but not in the opossums. In most cases the ileal strips of both species relaxed during electrical field stimulation, with tension returning to basal levels at the ends of the trains. This fall in basal tension during the stimulus train was seldom seen in the colonic strips of either species. All reponses to electrical field stimulation were abolished by tetrodotoxin (3×10^{-7} M) in the opossum and in the cat (3×10^{-6} M). In both species, the length-tension slopes were greatest just above the ileocecal junction where stimulus relaxation occurred. Tetrodotoxin (3×10^{-7} M) raised the basal tension in the ileal strips in both species and in the colonic strips only in the cat. The data indicate that the ileocecal junction muscle resembles that of the esophagogastric and gastroduodenal junctions in its relatively high degree of resistance to stretch and prominent neurogenic stimulus relaxation. However, in contrast to these other junctions, the ileocecal junction also possesses a tonic inhibitory innervation. The ileocecal junction appears to be characterized by a control system which differs from the systems of the ileum above and the colon below the junction.

3877 EFFECTS OF GASTROINTESTINAL HORMONES ON GASTROINTESTINAL MOTILITY AND ABSORPTION. *(Ger.)* Soergel, K. H. (Med. Coll. Wisconsin, Milwaukee). *Verh. Dtsch. Ges. Inn. Med.* 80:385-392, 1974.

3878 ELECTROMYOGRAPHIC STUDY OF GASTROINTESTINAL MOTILITY DURING VOMITING IN THE NONANESTHETIZED DOG. *(Fr.)* Monges, H.; Salducci, J.; Naudy, B. (Clin. Gastrointest. Dis. Nutr., Marseille, France). *Biol. Gastroenterol. (Paris)* 7(4):300, 1974.

3879 STUDY OF ELECTRICAL GASTROINTESTINAL ACTIVITY AFTER SURGERY IN MAN. *(Fr.)* Schang, J. C.; Dauchel, J.; Jaeck, D.; Grenier, J. F. (Munic. Hosp., Sgrasbourg, France). *Biol. Gastroenterol. (Paris)* 7(4):299, 1974.

3880 ENDOSCOPIC MEASUREMENTS OF PRESSURE WITH SEMICONDUCTORS: PRELIMINARY REPORT ON THE MEASURING TECHNIQUE. *(Ger.)* Vondrasek, P.; Eberhardt, G. (Westphal. Clin. Dig. Metab. Dis., Bad Driburg, Germany). *Z. Gastroenterol.* 12(6):453-458, 1974.

3881 EFFECTS OF PROSTAGLANDIN E_1 (PGE_1) ON ISOLATED STOMACH, SMALL INTESTINE, BRONCHI, AND UTERUS OF EXPERIMENTAL ANIMALS AND THE INTESTINAL EFFECTS OF THE AGENT IN VIVO. *(Jpn.)* Suzuki, T.; Hashikawa, T.; Takano, S.; Hayashi, A. (Fukushima Med. Coll., Japan). *Folia Pharmacol. Jpn.* 71:109-122, 1975.

3882 CHANGES OCCURRING IN THE MOTOR AND EVACUATORY ACTIVITY OF THE STOMACH, DUODENUM, AND GALLBLADDER DURING GRADUAL DEVELOPMENT OF PANCREATIC ATROPHY. *(Rus.)* Barashkova, G. M.; Troitskaia, V. B. (I. P. Pavlov Inst. Physiol., Leningrad, USSR). *Fiziol. Zh. SSSR* 61(4):610-614, 1975.

3883 EFFECT OF PROXIMAL GASTRIC VAGOTOMY AND TOTAL VAGOTOMY ON THE CO-ORDINATION OF ELECTRICAL ACTIVITY OF THE STOMACH AND DUODENUM. *(E.)* Aeberhard, P.; Bedi, B. S. (West. Infirm., Glasgow, Scotland). *Br. J. Surg.* 62(2):153, 1975.

3884 MOTOR AND ELECTRICAL ACTIVITY OF THE STOMACH AND JEJUNUM AFTER ADMINISTRATION OF ANGIOTENSIN II. *(Rus.)* Deleva, Zh. I.; Nikelov, N. A. (Med. Fac., Sofia, Bulgaria). *Fiziol. Zh. SSSR* 61(4):615-619, 1975.

3885 CHANGES IN GASTRIC MOTILITY PRODUCED BY NOCICEPTIVE STIMULATION OF THE SKIN IN RATS. *(E.)* Sato, A.; Sato, Y.; Shimada, F.; Torigata, Y. (Tokyo Metrop. Inst. Gerontol., Japan). *Brain Res.* 87(2/3):151-159, 1975.

3886 GASTRIC EMPTYING TESTS IN MAN. *(E.)* Sheiner, H. J. (Perth Med. Cent., Shenton Park, Australia). *Gut* 16(3):235-247, 1975.

3887 EFFECT OF 13-NORLEUCINE ANALOGUE OF MOTILIN ON STOMACH EMPTYING AND GASTRIC PEPSIN SECRETION IN MAN. *(Ger.)* Ruppin, H.; Domschke, S.; Domschke, W.; Wunsch, E.; Jaeger, E.; Demling, L. (Med. Clin., Univ. Erlangen, Germany). *Z. Gastroenterol.* 12(Suppl.):418, 1974.

3888 EFFECT OF GASTRIN AND SECRETIN ON STOMACH EMPTYING IN MAN. *(Ger.)* Meves, M.; Beger, H. G.; Huthwol, B. (Wastend Clin., Free Univ. Berlin, Germany). *Z. Gastroenterol.* 12(Suppl.):415-416, 1974.

3889 GASTRIC EMPTYING OF ORGANIC ACIDS IN THE DOG. *(E.)* Krejs, G. J.; Blum, A. L. (Triemli City Hosp., Zurich, Switzerland). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):63, 1975.

3890 EFFECT OF AZIDOMORPHINE AND RELATED SUBSTANCES ON THE INTESTINAL MOTILITY. *(E.)* Knoll, J.; Zseli, J.; Ronai, A.; Vizi, E. S. (Semmelweiss Univ. Med., Budapest, Hungary). *Pharmacology* 12(4/5):283-289, 1974.

3891 AN ANALYSIS OF THE PHENOMENON OF ACUTE TOLERANCE TO MORPHINE IN THE GUINEA-PIG ISOLATED ILEUM. *(E.)* Kosterlitz, H. W.; Waterfield, A. A. (Unit. Res. Addictive Drugs, Univ. Aberdeen, Scotland). *Br. J. Pharmacol.* 53(1):131-138, 1975.

3892 NEUROPHYSIOLOGY OF AUERBACH'S PLEXUS AND CONTROL OF INTESTINAL MOTILITY. *(E.)* Wood, J. D. (Univ. Kansas Med. Cent., Kansas City). *Physiol. Rev.* 35(2):307-324, 1975.

3893 RECORDING OF INTESTINAL MOTOR ACTIVITY WITH THE PERMANENT BALLOON IN CHRONIC EXPERIMENTS ON CATS. *(Rus.)* Poliakov, E. L.; Talan, M. I. (Pavlov Inst. Physiol., Acad. Sci., Leningrad, Russia). *Fiziol. Zh. SSSR* 61(2):305-306, 1975.

3894 THE IRREVERSIBLE BINDING OF ACETYLCHOLINE MUSTARD TO MUSCARINIC RECEPTORS IN INTESTINAL SMOOTH MUSCLE OF THE GUINEA-PIG. *(E.)* Robinson, D. A.; Taylor, J. G.; Young, J. M. (Med. Sch., Unov. Cambridge, England). *Br. J. Pharmacol.* 53(3):363-370, 1975.

3895 INTESTINAL SMOOTH MUSCLE FIBER: ELECTROPHYSIOLOGICAL AND PHARMACODYNAMIC STUDY. *(Fr.)* Salducci, J.; Remacle, J. P.; Monges, H. (no affil.). *Ann. Anesthesiol. Fr.* 15(Suppl. 1):14-17, 1974.

3896 COMPARITIVE ACTION OF BORIC, BENZENEBORONIC, AND p-METHYLBENZENEBORONIC ACIDS ON INTESTINAL SMOOTH MUSCLE. *(Fr.)* Chanh, P. H.; Sokan, I.; Quessada, M. H. (Inst. Physiol., Paul Sabatier Univ., Toulouse, France). *Agressologie* 15(2):131-136, 1974.

3897 CHOLINOCEPTIVE SITES IN THE GANGLIA OF MYENTERIC PLEXUS SUBSERVING THE PERISTALTIC REFLEX OF GUINEA-PIG ISOLATED ILEUM. *(E.)* Beleslin, D. B.; Malobabic, Z. S. (Med. Fac., Belgrade, Yugoslavia). *Neuropharmacology* 13(10/11): 1091-1094, 1974.

3898 ANTIPERISTALTIC INTESTINAL SEGMENTS: EXPERIMENTAL AND CLINICAL STUDY. *(Fr.)* Dauchel, J.; Schang, J. C.; Sava, G.; Jaeck, D.; Grenier, J. F. (no affil.). *Rev. Fr. Gastroenterol.* (102):63-64, 67-70, 1974.

3899 COLONIC AND SMALL INTESTINAL RESPONSE TO INTRAVENOUS PROSTAGLANDIN $F_{2\alpha}$ AND E_2 IN MAN. *(E.)* Milton-Thompson, G. J.; Cummings, J. H.; Newman, A.; Billings, J. A.; Misiewicz, J. J. (R. Nav. Hosp., Gosport, England). *Gut* 16(1):42-46, 1975.

3900 QUANTITATIVE INVESTIGATION OF INTESTINO-INTESTINAL REFLEXES. *(Rus.)* Groisman, S. D.; Dekush, P. G. (Inst. Physiol., Kiev Univ., USSR). *Patol. Fiziol. Eksp. Ter.* (3):51-55, 1974.

3901 STUDY OF THE DUODENAL PACEMAKER IN THE RAT. *(Fr.)* Santini, R.; Brard, E. (Dep. Physiol. Pharmacodyn., INSERM, Villeurbanne, France). *C. R. Soc. Biol.* [D] *(Paris)* 168(6/7):764-771, 1974.

3902 EXPERIMENTAL STUDY OF RETARDED INTESTINAL TRANSIT FOLLOWING EXTENSIVE RESECTIONS WITH PREILEAL TRANSPOSITION OF A SEGMENT OF THE COLON. *(It.)* Perisi, P.; Bianca, T.; Saba, V. (Inst. Clin. Gen. Surg. Surg. Ther., Univ. Sassari, Italy). *Chir. Patol. Sper.* 21(1):22-35, 1973.

3903 PHARMACOLOGY OF COLONIC MUSCLE. *(E.)* Bennett, A. (King's Coll. Hosp. Med. Sch., London, England). *Gut* 16(4):307-311, 1975.

3904 ELECTRICAL POTENTIAL DIFFERENCE OF COLONIC MUCOSA. *(E.)* Edmonds, C. J. (Univ. Coll. Hosp. Med. Sch., London, England). *Gut* 16(4):315-318, 1975.

3905 MECHANISM OF CHOLECYSTOKININ INHIBITION OF LOWER ESOPHAGEAL SPHINCTER PRESSURE. *(E.)* Fisher, R. S.; DiMarino, A. J.; Cohen, S. (Temple Univ. Health Sci. Cent., Philadelphia, Pa.). *Am. J. Physiol.* 228(5):1469-1473, 1975.

3906 STUDIES OF PARALYTIC ILEUS. EFFECTS OF INTRAPERITONEAL INJURY ON MOTILITY OF THE CANINE SMALL INTESTINE. *(E.)* Mishra, N. K.; Appert, H. E.; Howard, J. M. (Med. Coll. Ohio, Toledo). *Am. J. Surg.* 129(5):559-563, 1975.

3907 ANTAGONISM OF ADENOSINE 5'-TRIPHOSPHATE-INDUCED RELAXATION BY 2-2'-PYRIDYLISATOGEN IN THE TAENIA OF GUINEA-PIG CAECUM. *(E.)* Spedding, M.; Sweetman, A. J.; Weetman, D. F. (Sch. Pharm., Sunderland Polytech., Germany). *Br. J. Pharmacol.* 53(4):575-583, 1975.

3908 COLONIC MOTILITY. *(E.)* Misiewicz, J. J. (Cent. Middlesex Hosp., London, England). *Gut* 16(4):311-314, 1975.

3909 GASTROINTESTINAL MOTILITY: SOME ANATOMICAL AND PHYSIOLOGICAL ASPECTS IN THE INTACT ANIMAL AND AFTER PARTIAL GASTRECTOMY. *(E.)* Barrett, G. S. (no affil.). *Ann. R. Coll. Surg. Engl.* 56(5):247-248, 1975.

3910 SYMPOSIUM ON COLONIC FUNCTION. ELECTROPHYSIOLOGY OF THE COLON. *(E.)* Daniel, E. E. (Dep. Pharmacol., Univ. Alberta, Edmonton, Canada). *Gut* 16(4):298-306, 1975.

See also, 4377, 4506, 4530.

SECRETION AND METABOLISM

3911 THE INFLUENCE OF SYMPATHETIC ACTIVITY AND ISOPRENALINE ON THE SECRETION OF AMYLASE FROM THE HUMAN PAROTID GLAND. *(E.)* Speirs, R. L.; Herring, J.; Cooper, W. D.; Hardy, C. C.; Hind, C. R. K. (London Hosp. Med. Coll., England). *Arch. Oral Biol.* 19(9):747-752, 1974.

3912 EFFECT OF CATECHOLAMINE ANALOGS ON THE GLYCOGEN CONCENTRATION IN MOUSE PAROTID GLAND--RELATIONSHIP TO ADENOSINE 3',5'-MONOPHOSPHATE LEVELS AND INCREASED DEOXYRIBONUCLEIC ACID SYNTHESIS. *(E.)* Durham, J. P.; Butcher, F. R. (Dep. Biochem., Univ. Glasgow, Scotland). *Biochem. Pharmacol.* 24 (8):920-922, 1975.

3913 AGING AND ENZYMATIC CHANGES IN RAT PAROTID GLAND TISSUE. *(Ger.)* Chilla, R.; Arglebe, C.; Arold, R. (Otorhinolaryngol. Clin., Univ. Gottingen, Germany). *Acta Otorhinolaryngol. Belg.* 206 (4):311-317, 1974.

3914 ON RHYTHMICAL VARIATION OF THE PROTEIN CONTENT IN CELLS OF INTRALOBULAR DUCTS OF THE RAT PAROTID GLAND. *(Rus.)* Nechaeva, N. V.; Fateyeva, V. I. (Inst. Dev. Biol., USSR. Acad. Sci., Moscow). *Tsitologiya* 16(8):1033-1036, 1974.

3915 EFFECT OF VAGOTOMY AND PYLOROMYOTOMY ON THE ACTIVITY OF DUODENAL, COLONIC AND SPLENIC HYDROLYTIC ENZYMES IN RAT. *(Pol.)* Wesolowski, H.; Snop, S. (Inst. Histol. Embryol., Szcecin). *Pol. Tyg. Lek.* 29(48):2077-2080, 1974.

3916 ISOLATION AND IDENTIFICATION OF PROSTAGLANDIN E_2 FROM THE GASTROINTESTINAL TRACT OF SHARK Triakis scyllia. *(E.)* Ogata, H.; Nomura, T. (Lab. Marine Biochem., Tohoku Univ., Sendai, Japan). *Biochim. Biophys. Acta* 388(1):84-91, 1975.

3917 STUDIES ON DEGRADED CARRAGEENAN IN RATS AND GUINEA-PIGS. *(E.)* Grasso, P.; Gangolli, S. D.; Butterworth, K. R.; Wright, M. G. (Br. Indust. Biol. Res. Assoc., Carshalton, England). *Food Cosmet. Toxicol.* 13(2):195-201, 1975.

3918 EFFECT OF ISOPRENALINE INFUSION ON GASTROINTESTINAL SECRETION IN THE ANESTHETIZED RAT. INTERACTION WITH PROPRANOLOL. *(Fr.)* Dupont, C.; Jean, E.; Wepierre, J. (Pharm. Cent., Chatenay-Malabry, France). *Biol. Gastroenterol. (Paris)* 7(4): 298-299, 1974.

3919 CHEMOSENSITIVE VAGAL ENDINGS IN THE OESOPHAGUS OF THE CAT. *(E.)* Harding, R.; Titchen, D. A. (Dep. Physiol., Univ. Melbourne, Victoria, Australia). *J. Physiol. (Lond.)* 247(1):52P-53P, 1975.

See also, 3822.

SECRETION AND METABOLISM

Stomach

3920 EFFECTS OF PREDNISONE ON GASTRIC SECRETION AND DEVELOPMENT OF STOMACH LESIONS IN SWINE. *(E.)* Zamora, C. S.; Kowalczyk, T.; Hoekstra, W. G.; Grummer, R. H.; Will, J. A. (Dep. Vet. Sci., Univ. Wisconsin, Madison). *Am. J. Vet. Res.* 36(1): 33-39, 1975.

The effects of prednisone on ulceration and gastric secretion was studied in crossbred weanling pigs. The pigs were treated for 19 days with i.m. saline or i.m. prednisone (10, 20, 40, 80, or 160 mg/day); all animals were also treated i.m. with a long-acting antibiotic preparation. In the second experiment, the gastric contents were collected from five pigs with simple gastric fistulas both before and during treatment with prednisone (10 mg/kg, i.m.). In the first experiment, the prednisone-treated pigs developed pathologic changes in the glandular region of the stomach, primarily in the fundic area and occasionally in the pyloric and cardiac mucosas. The characteristic lesion was hemorrhagic gastritis, which was associated with massive gastric hemorrhages in the animals given the larger doses of prednisone. Epithelial changes were observed in the esophageal area, but there were no ulcers. In the animals with gastric fistulas, administration of prednisone for 8 days produced a significant increase in the volume and acidity of the gastric secretion. In addition, the hexosamine output, but not its concentration, increased significantly during the treatment period. Prednisone did not produce an immediate change in gastric secretion; there was a latent period of 1-2 days before marked changes were observed. Peak changes were seen around the third or fourth day, after which the acidity of the gastric secretion gradually decreased to pretreatment levels. The results indicate that although increased acidity may be involved in the development of lesions in the glandular region of the stomach, other factors probably also have a role in the pathogenesis of steroid-induced stomach lesions in swine.

3921 THE INFLUENCE OF FOOD CONSUMPTION AND RUNNING ACTIVITY ON THE ACTIVITY-STRESS ULCER IN THE RAT. *(E.)* Pare, W. P. (VA Hosp., Perry Point, Md.). *Am. J. Dig. Dis.* 20(3):262-273, 1975.

The extent to which food consumption and running activity contribute to the development of the activity-stress ulcer was studied in male ARS/Sprague-Dawley rats. In the first experiment, rats housed in activity cages were exposed to a daily 1-hr feeding schedule; control animals in standard cages were food yoked to the activity rats. A similar procedure was followed in the second experiment, in which both activity and control groups contained naturally active and less active animals, and in the third experiment, in which the rats' normal light-dark cycle was reversed. In the fourth experiment, rats representing three different age groups, and presumably three activity levels, were exposed to the activity-stress situation. In the first experiment, the rats housed in the activity cages died within 21 days and developed extensive ulcers in the glandular stomach; the food-yoked controls did not die and remained ulcer-free. Rats demonstrating high running activity levels under *ad libitum* feeding conditions subsequently developed a higher incidence of ulcers than low-activity rats when exposed to the activity-stress situation. At-

tempts to attenuate activity and lesion incidence by reversing the light-dark schedule failed, but a higher mortality level in rats under the reversed light-dark condition was related to higher activity levels in these animals. In the fourth experiment, young rats with a high activity levels died sooner and developed more ulcers than older rats with low activity levels. These results suggest that running activity, and not reduced food intake, was critically involved in the development of the activity-stress ulcers in these experiments.

3922 THE EFFECTS OF BURIMAMIDE AND METIAMIDE ON GASTRIC SECRETION IN THE CONSCIOUS CAT. *(E.)* Harris, D.; Smy, J. R.; Reed, J. D.; Venables, C. W. (Sch. Pharm., Sunderland Polytech., England). *Agents Actions* 4(5):311-313, 1974.

The effects of burimamide and metiamide on gastric acid secretion were studied in 10 cats with cannulated gastric fistulas. In four experiments, burimamide (2 mg/kg, i.v.), metiamide (4 mg/kg, i.m.), burimamide plus metiamide, and pentagastrin (6 µg/kg) were injected into the conscious animals; the gastric acid output was measured every 15 min. Burimamide significantly increased acid secretion over basal levels, the maximal increase occurring within 30 min of injection. Metiamide, on the other hand, had no significant effect on acid secretion. Although acid output was increased following the administration of burimamide plus metiamide, the response was significantly lower than that to burimamide alone. The total acid output in response to burimamide was only 18.5% of that observed in response to pentagastrin. These results suggest that burimamide has a partial agonistic action which is not shared by metiamide.

3923 BA^{++} ON THE RESTING FROG STOMACH: EFFECTS ON ELECTRICAL AND SECRETORY PARAMETERS. *(E.)* Rangachari, P. K. (Univ. California Sch. Med., San Francisco). *Am. J. Physiol.* 228(2):511-517, 1975.

The effects of Ba^{++} on the electrical and secretory activities of the resting frog stomach were studied. Gastric mucosae from bullfrogs were bathed in nutrient solutions containing potassium penicillin, streptomycin sulfate, KCl, histamine acetylcholine, NaSCN, and/or $BaCl_2$ (0.1-2.0 mM). Low chloride solutions were prepared using glucuronic acid titrated with NaOH to neutral pH. Ba^{++} added to the nutrient solution increased resistance, decreased the potential difference, and stimulated acid secretion in comparison to stomachs bathed in normal solutions. Under short-circuit conditions, the increase in the acid secretory rate was accompanied by a decrease in short-circuit current. These changes were reversed by NaSCN (10 mM), suggesting that Ba^{++} had not impaired the current-generating mechanism *per se*. Histamine-induced acid secretion was associated with an increase in net Cl^- flux, particularly the forward flux (N+S). Ba^{++} increased acid secretion with no increase in the forward flux, but with a decrease in the net Cl^- flux. Histamine, in the presence of Ba^{++} and low-Cl^- solutions, increased acid secretion and transmucosal resistance, suggesting the operation of a neutral pump in the secretion of HCl. The results suggest that Ba^{++} affects the movement of Cl^- across the tissue and induces a competition for Cl^- between the acid-secreting and current-generating mechanisms at the luminal surface.

3924 EFFECT OF ANTRECTOMY AND SUBSEQUENT VAGOTOMY ON THE SERUM GASTRIN RESPONSE TO FOOD IN DOGS. *(E.)* Booth, R. A. D.; Reeder, D. D.; Thompson, J. C. (Univ. Texas Med. Branch, Galveston). *Ann. Surg.* 181(2):191-195, 1975.

The food-stimulated release of extra-antral gastrin was studied in mongrel dogs with denervated fundic Heidenhain pouches. Serum gastrin concentrations and acid secretion from the Heidenhain pouches were measured after an 18-hr fast and every 15 min for 3 hr following the ingestion of a 200 g meat meal. The dogs were then subjected to antrectomy, with gastrointestinal continuity restored by means of a Billroth I gastroduodenal anastomosis or a Billroth II gastro-jejunostomy. The effect of food on serum gastrin and gastric acid secretion was again measured 3 weeks later. A few days later, some of the dogs were subjected to bilateral truncal vagotomy; the food studies were repeated 3 weeks afterward. Serum gastrin was measured by radioimmunoassay. In all dogs, antrectomy significantly depressed the mean basal levels of serum gastrin and abolished the rise in serum gastrin shown by intact animals in response to a meat meal. Food-induced pouch acid secretion was also significantly lowered by antrectomy. Vagotomy after antrectomy increased the basal levels of serum gastrin, but did not restore the serum gastrin response to a meal. The results suggest that biologically active forms of gastrin are released from the antrum in response to food, while biologically inactive basal levels of gastrin apparently originate from extra-antral sources. The post-vagotomy increase in basal (static) gastrin suggests vagal control of the metabolism of static, extra-antral gastrin.

3925 ELECTROPHYSIOLOGIC RESPONSES OF THE CANINE GASTRIC MUCOSA TO ENDOTOXIN, EPINEPHRINE, AND HEMORRHAGE. *(E.)* Chou, A. C.; Jacobson, E. D.; Shanbour, L. L. (Univ. Texas Med. Sch., Houston). *Circ. Shock.* 2(1):13-20, 1975.

The early electrophysiologic responses of an epithelial membrane were measured during endotoxin and hemorrhagic shock using an *in vivo* canine stomach preparation. A segment of the oxyntic gland area of the stomach was exposed in fasted mongrel dogs of both sexes. The potential difference across the stomach was monitored with a pair of calomel electrodes. In some animals, the LD_{100} dose of endotoxin (6 mg/kg) was given as a rapid i.v. bolus (group II) or by intra-arterial infusion (group III). In group IV, oligemic shock was induced by removal of blood from the femoral artery, and group V was given epinephrine (1 µg/kg/min) directly into the gastric artery for 10 min. In a second experiment, the effects of phentolamine (2.3 mg/kg given as 2 boluses and by continuous

infusion) on the potential difference after endotoxin was studied. In the third experiment, the effect of endotoxin and aminopyrine (given at 20 mg/kg loading dose and subsequent infusion at a rate of 5 mg/kg/hr) clearance from the gastric mucosal flap was measured. Given as a bolus, endotoxin caused an immediate decrease in the potential difference and an increase in the equivalent electrical resistance across the stomach; the same dose of endotoxin given as a continuous infusion produced no such changes. Oligemic shock and epinephrine had similar effects to those produced by the endotoxin bolus. The decrease in potential difference and increase in equivalent electrical resistance produced by endotoxin were blocked by phentolamine, and endotoxin produced an increase in the gastric mucosal blood flow as determined by aminopyrine clearance. The results suggest that the effect of endotoxin on the electrophysiology of the gastric mucosa is mediated primarily through the release of epinephrine, which acts directly on the active transport processes rather than through the induction of gastric mucosal ischemia.

3926 INTERACTION OF SECRETIN AND CARBAMINOYLCHOLINE ON GASTRIC SECRETION IN CATS. *(E.)* Vagne, M.; Perret, G. (INSERM, Edouard-Herriot Hosp., Lyon, France). *Digestion* 11(3/4):215-225, 1974.

Acid, pepsin, and gastric mucus secretion were studied during stimulation with a cholinergic, secretin, and a combination of both in three cats equipped with a gastric fistula and a Heidenhain pouch. Gastric secretion from both the gastric fistula and Heidenhain pouch was collected for three 1-hr periods following experiments using carbaminoylcholine (10 µg/kg for first hr, 20 µg/kg for second hr), synthetic secretin (0.5 U/kg for first hr, 3 U/kg for second hr), and a combination of these. Carbaminoylcholine slightly stimulated acid secretion and strongly stimulated pepsin and mucus secretion using determinations of uronic acid as representative of polysaccharides and fucose as representative of glycoproteins. Synthetic secretin induced a small stimulation of pepsin, uronic acid, and fucose secretions. Secretin added to cholinergic stimulation decreased the acid response and increased pepsin and mucus responses. The results suggest that, in cats, fundic mucus secretion is under both cholinergic and hormonal control.

3927 INHIBITION OF GASTRIC ACID SECRETION IN THE RAT BY ORALLY-ADMINISTERED SYNTHETIC PROSTAGLANDIN ANALOGUES: ENANTIOMORPHS OF 15-HYDROXY-15-METHYL-9-OXOPROSTANOIC ACID. *(E.)* Lippman, W. (Biochem. Pharmacol. Dep., Ayerst Lab., Montreal, Canada). *J. Pharm. Pharmacol.* 26(10):831-833, 1974.

The effects of orally-administered enantiomorphs of 15-hydroxy-15-methyl-9-oxoprostanoic acid (AY-22469) on basal gastric acid secretion and on the incidence of diarrhea were studied in mice and rats. The synthetic prostaglandin racemate AY-22469 inhibited basal gastric secretion with an ED_{50} of 4.3 mg/kg. The *ent*-isomer was similarly active with a parallel log dose-response curve, while the *nat*-isomer was 2.8-fold less effective than the *ent*-isomer with a parallel log dose-response curve. There was no effect on the volume of gastric juice produced. In mice, AY-22469 at a dose level up to 240 mg/kg caused diarrhea in 13% of the animals; at a similar dose level, the incidence of diarrhea was 25% with the *ent*- and *nat*-isomers. In rats, AY-22469 caused diarrhea with an ED_{50} of 120 mg/kg. The antisecretory activity of AY-22469 and the enantiomorphs appears to be due to their action on the acid concentration; cyclic adenosine-3',5'-monophosphate may be involved in this action. AY-22469 and its enantiomorphs may be useful therapeutic agents for the treatment of gastric hypersecretion and peptic ulcers.

3928 ALTERATIONS IN SECRETORY PATTERNS FOLLOWING ANTRECTOMY IN RATS WITH PAVLOV POUCHES. *(E.)* Lundell, L. (Inst. Physiol., Univ. Lund, Sweden). *J. Physiol. (Lond.)* 245(3):737-754, 1975.

The gastric secretory response to various stimuli was studied in conscious female Sprague-Dawley rats provided with Pavlov pouches. The antrum was also removed in some rats, gastrointestinal continuity being reestablished by an end-to-end gastroduodenostomy. The maximal acid secretory capacity was 60% lower in the antrectomized than in the intact Pavlov pouch rats 3 weeks after surgery; the difference increased with time as the maximal capacity of the intact animals increased. Antrectomy also reduced the interdigestive secretion of acid. Acid secretion in response to i.v. pentagastrin infusion (maximal dose 8 µg/hr for 6 hr) was reduced by about 50% 3 weeks after antrectomy and by about 65% after 6 weeks. The antrectomized and intact rats did not differ in this respect, however, when the responses were related to the maximal secretory capacity. The dose-response curve to paentagastrin revealed a reduced responsiveness to submaximal doses of this agent, following antrectomy. The maximal acid secretory response to i.v. histamine (15.625-2000 µg/hr) was also reduced after antrectomy, although the sensitivity to submaximal infusions of histamine appeared to be increased. The mean secretory output in response to 2-deoxy-D-glucose (50 mg/kg, i.v.) was reduced by about 65%, and that to food (2-3 gm standard pellet diet) was reduced by about 85% following antrectomy. After antrectomy, a background infusion of pentagastrin enhanced the secretory response to 2-deoxy-D-glucose and food, but did not restore the responses to the levels seen in the intact rats. The feeding responses as related to the maximal secretory capacity were, however, similar in both groups when pentagastrin was infused into the antrectomized animals. The interdigestive secretion of pepsin was reduced by about 60% after antrectomy, while the peak response to 2-deoxy-D-glucose was about twice the interdigestive level in both groups. The food-stimulated increase in pepsin secretion was of longer duration in the antrectomized rats. The results suggest that the reduction in maximal acid secretory capacity after antrectomy is due primarily to a parietal cell hypoplasia, and that the functional performance of

the parietal cells is altered after antrectomy to a degree that the response to methacholine plus histamine is not a true gauge of the maximal secretory capacity.

3929 DECREASED INCIDENCE OF INDOMETHACIN-INDUCED GASTRIC ULCERATION IN RATS BY BILE DUCT DIVERSION. *(E.)* Abtahi, F. S.; Djahanguiri, B. (Fac. Med., Univ. Tehran, Iran). *Br. J. Surg.* 62(2): 113-114, 1975.

The effect of bile drainage into different sites in the small bowel on the incidence of indomethacin-induced gastric ulceration was studied in male and female Sprague-Dawley rats. The fasted animals were left unoperated, sham-operated, or fitted with a cannula inserted into the bile duct; the other end of the cannula was inserted into the small bowel 0, 2.5, 5, 7.5, or 10 cm distal to the ampulla of Vater. The animals were then infused i.p. with saline or indomethacin (40 mg/kg) in saline solution. The stomachs were examined for signs of ulceration 4 hr later. Indomethacin produced ulceration in 100% of the unoperated and sham-operated animals. In the operated rats in which the cannula had been inserted 0, 2.5, 7.5, and 10 cm from the ampulla of Vater, indomethacin produced 100, 80, 50, 30, and 10% ulceration, resp. The incidence of indomethacin-induced ulceration was significantly reduced in the latter three groups of operated rats compared with the unoperated, sham-operated, and operated rats in whom the cannula was inserted 0 cm from the ampulla of Vater. The results show that the exclusion of bile reflux from the stomach prevents the appearance of indomethacin-induced gastric ulceration in fasted rats. They suggest that bile reflux plays an as yet undetermined role in the pathogenesis of indomethacin-induced gastric ulceration in rats.

3930 LIPOATE EFFECT ON CARBOHYDRATE AND LIPID METABOLISM AND GASTRIC H^+ SECRETION. *(E.)* Harris, J. B.; Alonso, D.; Park, O. H.; Cornfield, D.; Chacin, J. (San Francisco Gen. Hosp., Calif.). *Am. J. Physiol.* 228(3):964-971, 1975.

Some of the factors regulating the use of carbohydrates and lipids as energy sources by frog gastric mucosa were examined. Acid secretion and oxygen consumption were measured in the gastric mucosa of *Rana catesbeiana* following treatment with pyruvate, thiamine, thiamine pyrophosphate, lipoate, glucose, lactate, histamine, and/or various fatty acids. Both acid secretion and oxygen consumption by the mucosae were sharply stimulated by lipoate; this was followed by a rapid decline to below control values. Glucose or lactate alone had no effect on either parameter, and pyruvate alone caused only a slight stimulation of oxygen consumption. However, treatment of lipoate-treated mucosae with any of these compounds increased the stimulatory effect of the lipoate and markedly slowed the rate of decline subsequent to maximum stimulation. Various fatty acids had a moderate-to-large stimulatory effect on both acid secretion and oxygen consumption. However, lipoate pretreatment decreased the stimulatory effect of butyrate by 56%, that of decanoate by 87%, and that of palmitate by 60%; propionate became an inhibitor in the presence of lipoate. Lipoate also increased oxidation of exogenously supplied fatty acids compared to the levels seen in the absence of exogenous substrate. Lipoate-treated mucosae did not respond to histamine, but the addition of glucose restored responsiveness. The results indicate that the β-oxidation of fatty acids plays a major role in the acid secretory process and that it is centrally involved in the previously reported stimulation of acid secretion by cyclic AMP and histamine.

3931 CHEMICAL STIMULATORY MECHANISM IN GASTRIC SECRETION. *(E.)* Cieszkowski, M.; Konturek, S. J.; Obtulowicz, W.; Tasler, J. (Med. Acad., Krakow, Poland). *J. Physiol. (Lond.)* 246(1):143-157, 1975.

The effects of bovine serum albumin, pepsin-digested albumin, an amino acid mixture, liver extract, and mannitol (control) on the serum gastrin level, gastric mucosal blood flow, and acid secretion from the Heidenhain pouch were measured in three mongrel dogs. Distention of the Heidenhain pouch with mannitol or albumin (particularly pepsin-digested albumin) at pH 5.0 produced similar pressure-related increases in acid secretion, reaching a peak of only 10% of the maximal response to histamine. The highest acid output (about 80% of the maximal response to histamine) was obtained with liver extract; an amino acid mixture containing all essential amino acids stimulated acid secretion to a lesser degree. In no case did the secretions from gastric or pancreatic fistulas or the serum gastrin level change from basal values. I.v. atropine (100 μg/kg/hr) and irrigation of the pouch with 50 ml of 0.1% atropine or 2% xylocaine solution significantly depressed the pouch response to liver extract; irrigation with 0.5% acetylcholine did not affect the response, and i.v. urecholine (10 μg/kg/hr) increased the response. The pouch response to chemical stimulation by peptic digests was greatly potentiated by combining this with distention of the oxyntic gland area. When the pH of the liver extract in the Heidenhain pouch was gradually decreased from 5.0 to 1.0, there was a concomitant decrease in acid secretion and in the mucosal blood flow, both falling to the basal level at pH 1.0. Exogenous secretin given in graded doses from 0.5-8.0 U/kg/hr caused a small, dose-related inhibition of the acid response to liver extract and an increase in the mucosal blood flow; there was no significant change in the serum gastrin level. The results suggest that the products of protein digestion, polypeptides and free amino acids, are potent chemical stimulants of acid secretion from the oxyntic gland area. The stimulation of this area by peptic digests induces acid secretion by a local, gastrin-independent, partially neural reflex mechanism which is sensitive to pH, pressure, and secretin.

3932 CIRCADIAN PLASMA GASTRIN PATTERNS IN FEEDING AND FASTING MAN. *(E.)* Moore, J. G.;

Wolfe, M. (Univ. Utah Coll. Med., Salt Lake City). *Digestion* 11(3/4):226-231, 1974.

The effects of feeding and fasting on the circadian pattern of plasma gastrin concentrations were studied in human volunteers. Basal gastric acid collections were obtained every 24-hr from healthy male adults. The hourly secretory pattern, which described a circadian rhythm, was compared to serial plasma gastrin levels in subjects who underwent 24-hr gastric intubation and aspiration, subjects who ate and exercised freely, and subjects who fasted but were unintubated. A circadian rhythm of plasma gastrin levels was observed only in subjects who ate and exercised freely. The results confirm previous findings that the circadian pattern of gastric acid secretion is unrelated to plasma gastrin levels and support the belief that the circadian rhythm of plasma gastrin levels is a result of the effects of feeding on antral gastrin release.

3933 PENTAGASTRIN INCREASES PHENYLALANINE AND DECREASES HISTADINE INCORPORATION INTO PROTEINS OF RAT STOMACH. *(E.)* Evans, D. C.; Lin, T. M. (Res. Lab., Eli Lilly Co., Indianapolis, Indiana). *Res. Commun. Chem. Pathol. Pharmacol.* 10(3):597-600, 1975.

The action of pentagastrin on the incorporation of [^{14}C] L-phenylalanine and [^{14}C] L-histidine into the stomach and duodenum proteins of rats was studied. Sixty female rats were treated with [^{14}C] L-phenylalanine or [^{14}C] L-histidine (20 µCi/kg, i.p.) 1 hr before receiving pentagastrin (250 µg/kg, s.c.). One hr after injection of pentagastrin the incorporation of [^{14}C] L-phenylalanine into the proteins of the squamous and fundic portions of the stomach was significantly increased by 18% and 17.5%, resp. The uptake of [^{14}C] L-histidine into the proteins was significantly decreased in the squamous region (-24%), fundic region (-18%), and duodenal region (-16%). Incorporation of histidine and phenylalanine into the proteins of the pylorus was not significantly affected. The results support the view that gastrin plays a dual role on gastric function: a trophic action to enhance protein synthesis; and a stimulating action on histidine metabolism favoring histamine production.

3934 DOES HISTAMINE HAVE TWO VASODILATOR ACTIONS ON THE GASTRIC MUCOSA? *(E.)* Reed, J. D.; Sanders, D. J. (Med. Sch., Univ. Newcastle upon Tyne, Engalnd). *Agents Actions* 5(1):1-3, 1975.

Oxygen consumption and extraction were measured during gastric stimulation with histamine and gastrin pentapeptide to determine whether there is an extra mucosal blood flow component in addition to that linked to parietal cell activity. Gastric activity was stimulated by histamine (45 µg/kg, i.v.) in three cats and by pentapeptide (2 µg/min) in five. Samples of gastric venous blood were taken at the rate of 1 ml/min; hemoglobin concentration and % oxygen saturation were measured. Steady state conditions were ensured by taking values obtained between 30 and 60 min from start of stimulation. During gastrin pentapeptide stimulation, there was significantly less mucosal blood flow/U H^+ compared to the histamine responses. Oxygen consumption was dependent upon acid secretion in a similar way for both histamine and gastrin stimulation. The degree of oxygen extraction from the gastric circulation was significantly less during histamine stimulation than during gastrin stimulation. The results support the hypothesis that histamine causes a vasodilatation in the gastric mucosa in two ways, a direct dilator action and one associated with secretory activity.

3935 PEPSIN OUTPUT AFTER DISRUPTION OF THE HUMAN GASTRIC MUCOSAL BARRIER BY TAUROCHOLIC ACID. *(E.)* Ivey, K. J.; Parsons, C. (R. Prince Alfred Hosp., Sydney, Australia). *Am. J. Dig. Dis.* 20(4):362-364, 1975.

Pepsin output in man was studied after the gastric mucosal barrier was altered by taurocholic acid. Test solutions containing 10 mM taurocholic acid in 160 mM HCl were instilled into the stomachs of five healthy volunteers. Significant back diffusion of H^+ and exsorption of Na^+ occurred, but there was no significant increase in pepsin output compared to control studies using HCl alone. The results show that 160 mM HCl increases pepsin output in man, but altering the gastric mucosal barrier with taurocholic acid in 160 mM HCl does not produce any greater output of pepsin.

3936 EVIDENCE FOR OXYNTOPYLORIC REFLEX FOR RELEASE OF ANTRAL GASTRIN. *(E.)* Debas, H. T.; Walsh, J. H.; Grossman, M. I. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gastroenterology* 68(4):687-690, 1975.

The effect of distention of the vagally innervated oxyntic gland area on the release of gastrin from the vagally innervated pyloric gland area was studied in dogs. Three dogs were prepared with vagally innervated pouches of both the oxyntic gland area and the pyloric gland area (antrum). When the antral pouch was filled but not distended with 0.1 M $NaHCO_3$, graded distention of the oxyntic pouch caused graded increases in serum gastrin and graded increases in acid secretion from the oxyntic pouch. When the antral pouch was filled with 0.1 M HCl, graded distention of the oxyntic pouch evoked no increase in serum gastrin and the acid responses from the oxyntic pouch were much smaller than when the antrum was alkaline. The results support the hypothesis that distention of the oxyntic gland area initiates an oxyntopyloric reflex that causes release of gastrin from the pyloric gland area.

3937 EFFECTS OF INTRADUODENAL AMINO ACIDS, FATTY ACIDS, AND SUGARS ON SECRETIN CONCENTRATIONS. *(E.)* Boden, G.; Essa, N.; Owen, O. E. (Temple Univ. Health Sci. Cent., Phila., Pa.). *Gastroenterology* 68(4):722-727, 1975.

The effects of amino acids, fatty acids, and iso- and hypertonic solutions of sugars on serum concentrations of immunoreactive secretin (IRS) were studied in 21 anesthetized dogs. The test substances, an 8.5% amino acid mixture, a 50 mM Na oleate solution, an oleic acid bile mixture, 5% fructose, 40% sucrose, and 9% NaCl solutions were infused intraduodenally at constant rates. HCl (160 mM) was infused at the end of most experiments to demonstrate responsiveness of the animals. Infusion of amino acids, Na oleate, the oleic acid bile mixture, and 5% fructose all failed to significantly change portal or femoral venous IRS concentrations. NaCl (9%) caused a slight depression; 40% sucrose caused a slight increase in IRS concentrations; however, neither change was statistically significant. HCl significantly increased portal and femoral venous IRS concentration and was the only substance that increased pancreatic flow and bicarbonate excretion. The results suggest that oleic acid, amino acids, fructose, and hypermolar solutions at the concentrations tested have no major effect on IRS release; HCl is the only physiological substance shown to reproducibly promote secretin secretion.

3938 THE EFFECT OF CARBENOXOLONE ON CHANGES IN CANINE AND HUMAN GASTRIC MUCOSA CAUSED BY TAUROCHOLATE AND ETHANOL. *(E.)* Gordon, M. J.; O'Brien, P.; Skillman, J. J.; Silen, W. (Harvard Med. Sch., Boston, Mass.). *Surgery* 77(5):707-714, 1975.

The effect of carbenoxolone on gastric mucosal permeability was studied in canine gastric pouches and in healthy human subjects; its effect on ethanol- and taurocholate-induced changes was also determined. Three dogs were prepared with an antrectomy and a vagally denervated gastric pouch. Perfusion of the canine pouches with sodium taurocholate (40 mM) was associated with an increase in H^+ back diffusion. The effect was not diminished by 10 days of carbenoxolone treatment. A significant increase of gastric mucosal permeability was observed in six normal human subjects after instillation of ethanol (20% v/v). After 3 weeks of oral ingestion of carbenoxolone, there was inconsistent protection against ethanol-induced increases in gastric mucosal permeability. Basal secretion of H^+ and post-ethanol H^+ secretion appeared to be diminished by carbenoxolone. The results suggest that carbenoxolone does not protect against taurocholate- and ethanol-induced increases in gastric mucosal permeability in the dog or in man and that its beneficial effect on gastric ulcer is by a mechanism other than prevention of H^+ back diffusion.

3939 THE ROLE OF BILE REFLUX IN THE DEVELOPMENT OF COLD-RESTRAINT GASTRIC LESIONS. *(E.)* Meeroff, J. C.; Paulsen, G.; Guth, P. H. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Am. J. Dig. Dis.* 20(4):365-369, 1975.

The role of bile reflux in the development of gastric mucosal lesions during cold-restraint stress was studied in the rat. Sprague-Dawley rats (40) were randomized into four groups: one underwent sham operations with no cold-restraint stress; the other three were subjected to cold-restraint stress for 3 hr following sham operations, pyloric ligation, or bile duct ligation. The stomachs were then removed and the severity of lesions recorded; hematocrit readings were made on blood from the hearts. The mean lesion scores of all groups exposed to cold-restraint stress were similar but much higher than those of the unrestrained sham-operated group. Mean hematocrit readings in all groups exposed to cold-restraint stress were lower than in the control group. There was a moderate correlation between severity of mucosal lesions and hematocrit reading. The results suggest that bile reflux is not necessary for formation of cold-restraint stress lesions in the rat and that hematocrit readings may be useful in measuring blood loss secondary to experimental mucosal lesions.

3940 SIGNIFICANCE OF LOCAL CIRCULATORY DISTURBANCE IN THE EXPERIMENTAL PRODUCTION OF GASTRIC ULCER. *(E.)* Sekine, T.; Shiratori, T. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 114(2):121-130, 1974.

The relation between experimental production of gastric ulcer and local circulatory disturbance was investigated in dogs by ligating gastric vessels accompanied with or without histamine injection. Dogs (62) were divided into five groups and the following observations made in each group: macroscopic findings of the gastric mucosa; microscopic findings of ulcer; incidence of production of ulcers; and acid secretory response to histamine. Gastric ulcers were similar to those seen in humans except for little regeneration of mucosa or proliferation of connective tissue at the base and margin of the ulcer. The incidence of gastric ulcers was extremely high in groups having had both histamine administration (50 mg histamine dihydrochloride in beeswax, i.m.) and gastric vessel ligation compared with groups having had only histamine. Gastric acid secretion in response to histamine also showed a fall in free acidity and a marked decrease in volume after ligation of gastric vessels. The results suggest that decrease in mucosal resistance induced by ligation of gastric vessels, rather than gastric acid secretion, may play an important role as an etiologic factor in the production of gastric ulcers.

3941 EFFECT OF HYPNOTIC SUGGESTION OF RELAXATION ON BASAL AND BETAZOLE-STIMULATED GASTRIC ACID SECRETION. *(E.)* Stacher, G.; Berner, P.; Naske, R.; Schuster, P.; Bauer, P.; Starker, H.; Schulze, D. (Psychiatric 1st Surg. Clin., Univ. Vienna, Austria). *Gastroenterology* 68(4):656-661, 1975.

The effect of relaxation suggestion in hypnosis on basal and betazole stimulated gastric acid secretion was examined in four healthy female volunteers (21-24 yr-old) susceptible to hypnosis. Acid output was measured by intragastric titration and a pH-sensitive telemetry capsule. Three series of experiments, each lasting 3 hr, were carried out: 1) after 1 basal hr,

administration of 1.7 mg/kg betazole s.c.; 2) after the basal hr, administration of saline s.c. and induction of hypnosis; relaxation was suggested for 1 hr, after which posthypnotic amnesia was suggested and the subject awakened; and 3) procedure as in experiment 2 but 1.7 mg/kg betazole s.c. instead of saline. Each subject underwent two of each series. A completely crossed 4-factorial analysis of variance, accounting for treatment, time, replication, and subject factors was performed. Suggestion of relaxation in hypnosis resulted in a significant reduction of unstimulated (2.16 mEq/15 min) as well as betazole-stimulated (0.62 mEq/15 min) acid secretion and in a decrease of gastric motility. Both effects were reversed after the termination of hypnosis. It is suggested that these effects originate in the central nervous system and are mediated *via* an alteration of the stimulating and/or inhibiting tone of the autonomic nervous system.

3942 GASTRINS IN HUMAN ANTRUM, DUODENUM AND PERIPHERAL CIRCULATION. *(E.)* Vinik, A. I.; Grant, B. J.; Novis, B. (Dep. Med., Univ. Cape Town, South Africa). *S. Afr. Med. J.* 49(8):255-257, 1975.

The nature of the gastrins extracted from human antrum, duodenum, and peripheral circulation was studied using blood samples and biopsy specimens from 3 patients. The major form of gastrin found in the pyloric antrum was the heptadecapeptide form; the duodenum contained primarily high-molecular weight gastrin (MW 3,900) and almost no heptadecapeptide gastrin. Fasting serum samples contained mostly high-molecular weight gastrin. The results, in conjunction with previous observations that arginine-stimulated gastrin is comprised of mostly the heptadecapeptide and the lower molecular weight forms, suggest that stimulated gastrin is derived mainly from the pyloric antrum and that the large molecular series are produced by G cells; specific local cleavage processes then determine the nature of gastrin released.

3943 THE INFLUENCE OF HYPERCALCEMIA ON GASTRIC SECRETION AND SERUM GASTRIN CONCENTRATION IN INTACT AND THYRO-PARATHYROIDECTOMIZED RATS. *(E.)* Johansson, H.; Krause, U.; Nilsson, F.; Lundqvist, G. (Univ. Hosp., Uppsala, Sweden). *Ups. J. Med. Sci.* 79(3):161-163, 1974.

Gastric acidity and serum gastrin concentration were measured in thyro-parathyroidectomized (14) and sham-operated (14) rats after calcium infusion. Calcium was given in a dose of 2.6 mg/100 g in 0.5 ml administered by continuous infusion at 0.1 ml/min. Plasma gastrin and calcium concentrations were determined 15, 30, 60, 90, and 120 min after the start of the calcium infusion. Gastric juice was collected between 0-2 and 2-4 hr. A moderate H^+ decrease, and unchanged Cl^- concentration and plasma gastrin level were demonstrated in both experimental groups. Although calcium may increase the release of gastrin in man, the results of these experiments showing no change in the serum gastrin concentration after calcium infusion led to the conclusion that such a mechanism does not exist in the rat.

3944 DIRECT INHIBITION OF GASTRIC ACID AND PEPSIN SECRETION BY GROWTH-HORMONE RELEASE-INHIBITING HORMONE IN CATS. *(E.)* Gomez-Pan, A.; Reed, J. D.; Albinus, M.; Shaw, B.; Hall, R.; Besser, G. M.; Coy, D. H.; Kastin, A. J.; Schally, A. V. (Dep. Med., Univ. Newcastle upon Tyne, England). *Lancet* 1(7912):888-890, 1975.

The inhibitory action of growth-hormone release-inhibiting hormone on acid and pepsin secretion stimulated by pentagastrin was studied in six conscious cats prepared with gastric fistulas at least 1 yr earlier and compared with the inhibition of food-stimulated acid secretion. On separate days each animal was infused for 60 min with growth-hormone release-inhibiting hormone (10 µg/kg/min) starting 15 min before the s.c. injection of pentagastrin (6 µg/kg) or the ingestion of food, and continuing for 45 min. Gastric juice was collected throughout the period and for 75 min. Growth-hormone release-inhibiting hormone inhibited gastric acid and pepsin secretion in response to pentagastrin and food stimulation in cats. This effect was mediated by a direct action on the parietal and peptic cells. No evidence of a post-inhibitory acid or pepsin secretory rebound was obtained. A compound of hypothalamic origin, then, can exert a direct effect on exocrine secretion. It is suggested, in view of its widespread actions and its presence in relatively high concentrations in various organs apart from the hypothalamus, that growth-hormone release-inhibiting hormone, in addition to its action as a hypothalamic regulatory hormone, might also be responsible for local coordination of endocrine and exocrine secretion.

3945 DIRECT INHIBITION OF GASTRIC ACID BY GROWTH-HORMONE RELEASE-INHIBITING HORMONE IN DOGS. *(E.)* Barros D'Sa, A. A. J.; Bloom, S. R.; Baron, J. H. (R. Postgrad. Med. Sch., London, England). *Lancet* 1(7912):886-887, 1975.

To determine if growth-hormone release-inhibiting hormone has any direct action on gastric acid production independent of its effect on gastrin release, cyclic growth-hormone release-inhibiting hormone was infused at a rate of 20 µg/kg/hr into four dogs with gastric fistulas. Gastric acid output fell sharply in response to three stimuli of acid production: a meal; and pentagastrin (0.1 µg/kg/hr) and histamine (0.5 µg/kg/hr) infusions. During a 2-hr infusion of growth-hormone release-inhibiting hormone after a meat meal, gastric acid was totally inhibited in each dog with some return of secretion after stopping the infusion. Pentagastrin-stimulated acid production was inhibited 97% by growth-hormone release-inhibiting hormone infusion and histamine-stimulated acid secretion was inhibited 71%. Recovery of acid secretion to almost pre-growth-hormone release-inhibiting hormone levels was seen by the end of the 3rd hr. The fact that acid output was inhibited during pentagastrin administration suggests

that growth-hormone release-inhibiting hormone has a direct effect on the gastric parietal cell.

3946 INTESTINAL PHASE OF GASTRIC ACID SECRETION: AUGMENTATION OF MAXIMAL RESPONSE OF HEIDENHAIN POUCH TO GASTRIN AND HISTAMINE. *(E.)* Debas, H. T.; Slaff, G. F.; Grossman, M. I. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gastroenterology* 68(4):691-698, 1975.

A study was conducted to determine whether serum gastrin increases during the intestinal phase of gastric acid secretion, to investigate the interactions of the intestinal phase with histamine and with endogenous and exogenous gastrin, and to examine the effect of vagotomy on these interactions. In four antrectomized dogs with gastric fistulas and Heidenhain pouches, a liver extract solution of $NaHCO_3$ as control was infused into the gastric fistula for 4 hr. During the last 2 hr, a maximal dose of either pentagastrin (8 µg/kg/hr) or histamine (80 µg/kg/hr), or saline as control, was given i.v. The studies were repeated after truncal vagotomy. Liver extract alone caused a small Heidenhain pouch response but produced no rise in serum immunoreactive gastrin. Intestinal perfusion with liver extract augmented the maximal Heidenhain pouch response to pentagastrin three-fold before and two-fold after vagotomy. The maximal Heidenhain response to histamine was augmented 1 1/2 fold both before and after vagotomy. In another group of three dogs provided with antral pouches, gastric fistulas, and Heidenhain pouches, infusion of liver extract into the gastric fistula produced a similar augmentation of the Heidenhain pouch maximal response to endogenous gastrin released by antral distention with liver extract. It is concluded that liver extract releases enterooxyntin from the intestine, a hormone capable of augmenting the maximal Heidenhain pouch responses to both gastrin and histamine, that this hormone is distinct from gastrin, that vagotomy does not abolish its release, and that its release by food in the intestine accounts for the greater maximal Heidenhain pouch response to a meal than to exogenous gastrin.

3947 MODEL FOR ISOLATED GASTRIC CELLS TO STUDY THE ENZYMATIC MECHANISM INVOLVED IN ACID SECRETION. *(Fr.)* Lewin, M.; Cheret, A. M.; Grelac, F.; Soumarmon, A.; Ghesquier, D.; Girodet, J. (Bichat Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4):295-296, 1974.

3948 DIALYSIS STUDIED OF GASTRIC JUICE. *(Rus.)* Rogozina, E. M.; Ryss, E. S.; Dikaia, L. F. (Leningrad Sci. Res. Inst. Radiol. Hyg., USSR). *Lab. Delo* (3):135-136, 1975.

3949 GASTRIC ACID SECRETION IN THE LIZARD. IONIC REQUIREMENTS AND EFFECTS OF INHIBITORS. *(E.)* Hansen, T.; Slegers, J. F. G.; Bonting, S. L. (Dep. Physiol. Biochem., Univ. Nijmegen, Netherlands). *Biochim. Biophys. Acta* 382(4):590-608, 1975.

3950 A METABOLIC BASIS FOR INEFFICIENT HCl SECRETION. *(E.)* Sernka, T. J. (Louisiana State Univ. Med. Cent., Shreveport). *Physiol. Chem. Phys.* 7(1):77-78, 1975.

3951 COMPARITIVE STUDIES OF PEPSIN AND ACID SECRETION IN RATS GIVEN VARIOUS GASTROINTESTINAL HORMONES. *(Ger.)* Schmidt-Wilcke, H. A.; Nawrath, G. D. (St. Franzikus Hosp., Munster, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:528-530, 1974.

3952 HORMONAL INHIBITION OF GASTRIC AND PANCREATIC SECRETION. *(Ger.)* Dotevall, G. (Med. Clin., Univ. Goteborg, Sweden). *Verh. Dtsch. Ges. Inn. Med.* 80:380-385, 1974.

3953 INHIBITION OF GASTRIC SECRETION IN MAN BY ORAL ADMINISTRATION OF CALCITONIN. *(Ger.)* Hotz, J.; Goebell, H.; Minne, H.; Ziegler, R. (Cent. Intern. Med. Pediatr., Univ. Ulm, Germany). *Z. Gastroenterol.* 12(Suppl.):420-421, 1974.

3954 CALCITONIN AND GASTRIC SECRETION: THE DIFFERENT EFFECTS OF CALCITONIN ON GASTRIC ACID AND PEPSIN SECRETION DURING STIMULATION WITH PEPTAGASTRIN, BETAZOLE, AND INSULIN HYPOGLYCEMIA. *(Ger.)* Hotz, J.; Goebell, H.; Minne, H.; Ziegler, R. (Dep. Intern. Med., Univ. Ulm, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:523-526, 1974.

3955 BLOOD PRESSURE AND HEART RATE AFTER MAXIMAL STIMULATION OF GASTRIC SECRETION WITH PENTAGASTRIN IN SUBJECTS WITH NORMAL BLOOD PRESSURES. *(Ger.)* Merguet, P.; Ewers, H. R.; Brouwers, H. P. (Med. Clin., Univ. Essen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:561-564, 1974.

3956 CHOLINERGIC SENSITIZATION OF THE OXYNTIC GLANDS TO GASTRIN. *(Fr.)* Dubrasquet, M.; Svensson, S. O.; Emase, S. (Karolinska Hosp., Stockholm, Sweden). *Biol. Gastroenterol. (Paris)* 7(4): 294-295, 1974.

3957 EFFECTS OF ANTICHOLINERGIC AGENTS ON GASTRIC SECRETION STIMULATED BY PENTAGASTRIN INFUSION. *(E.)* Laudano, O. M.; Aparicio, N. J. (Centenario Hosp., Rosario Med. Sch., Argentina). *Tijdschr. Gastroenterol.* 17(1):7-16, 1974.

3958 THE EFFECT OF HISTAMINE H_2-RECEPTOR BLOCKADE WITH METIAMIDE ON SERUM GASTRIN LEVELS IN MAN. *(E.)* Barbezat, G. O.; Grant, B.; Bank, S.; Vinik, A. (Dep. Med., Univ. Capetown, South Africa). *Gut* 16(3):186-187, 1975.

3959 ISOLATION OF PEPSIN FROM GASTRIC JUICE OF DOGS. *(Ukr.)* Matsuy, S. P.; Svystun, T. I.; Zahorodneva, A. H.; Guly, M. F. (A. V. Palladin Inst. Biochem., Kiev, USSR). *Ukr. Biokhim. Zh.* 46 (6):782-784, 1974.

3960 FUNCTIONAL CONDITION OF THE STOMACH AND ITS GASTRIN CELLS. *(Rus.)* Buletsa, V. I,; Zheltvai, V. V.; Langazo, Z. P. (Uzhgorod Branch, Odessa Inst. Balneother., USSR). *Fiziol. Ah. SSSR* 61(2):298-300, 1975.

3961 REGULATION OF GASTRIC AND EXTRAGASTRIC RELEASE OF GASTRIN. *(Ger.)* Hausamen, T. U.; Fritsch, W. P.; Rick, W.; Kleybrink, H.; Schact, U. (1st Med. Clin A, Univ. Dusseldorf, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:518-520, 1974.

3962 CHANGES IN THE LEVEL OF BLOOD GASTRIN AFTER THE REMOVAL OF THE ANTRAL MUCOSA. *(Rus.)* Erspamer, V.; Melchiorri, P.; Soprantci, Ch.; Impeta, D.; Rosova, E. I. (Inst. Med. Pharm., Univ. Rome, Italy). *Fiziol. Zh. SSSR* 61(3):433-436, 1975.

3963 TISSUE UPTAKE OF LABELED GASTRIN (^{125}I OR ^{3}H) IN THE GASTRECTOMIZED RAT. *(Fr.)* Dubrasquet, M.; Millat, B. Accary, J. P.; Gislon, J.; Morgat, J. L.; Girma, J. P. (Bichat Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4):298, 1974.

3964 KINETIC ANALYSIS OF THE EFFECT OF GASTRIN ON THE PROLIFERATION OF PARIETAL AND CHIEF CELLS. *(Fr.)* Willems, G.; Lehy, T. (Bichat Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4): 296-297, 1974.

3965 INHIBITION OF ACID AND GASTRIN RESPONSE TO FEEDING BY ATROPINE IN CATS. *(E.)* Emas, S.; Svensson, S. O.; Dorner, M.; Kaess, H. (Karolinska Hosp., Stockholm, Sweden). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):59-60, 1975.

3966 THE SIGNIFICANCE OF THE APPEARANCE OF GASTRIN IN ANTRAL PERFUSATES FROM DOGS. *(E.)* Andersson, S. Nilsson, G. (Dep. Pharmacol., Karolinska Inst., Stockholm, Sweden). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):64, 1975.

3967 THE EFFECT OF 15 (R) 15 METHYL PGE_2 METHYL ESTER ON SERUM GASTRIN LEVELS IN MAN. *(E.)* Adaikan, G. P.; Salmon, J. A.; Karim, S. M. M. (Kandang Kerbau Hosp., Univ. Singapore). *J. Int. Res. Commun.* 2(9):1587, 1974.

3968 DOUBTFUL ROLL [SIC!] OF ENDOGENOUS GASTRIN IN CHICKEN GASTRIC SECRETION BY VAGAL STIMULATION. *(E.)* Kokue, E.; Hayama, T. (Dep. Vet. Sci., Fac. Agric., Tokyo Univ. Agric. Technol., Japan). *Experientia* 31(2):197-198, 1975.

3969 ABO BLOOD GROUPS AND SERUM GASTRIN. *(E.)* Mazzacca, G.; Budillon, G.; De Marco, F.; Carrella, M.; Cimino, L. (2nd Sch. Med., Univ. Naples, Italy). *Acta Hepatogastroenterol. (Stuttg.)* 22(1): 44-46, 1975.

3970 ADENYLATE CYCLASE AND PHOSPHODIESTERASE IN RAT GASTRIC MUCOSA. *(E.)* Ruoff, H. J.; Sewing, K. F. (Dep. Pharmacol., Univ. Tubingen, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 22(1): 63-64, 1975.

3971 CHANGES IN ACETYLCHOLINESTERASE ACTIVITY IN THE STOMACH OF A VAGOTOMIZED RAT. *(It.)* Lise, M.; Perrino, G.; Cordioli, G. P. (Inst. Clin. Gen. Surg. Surg. Ther., Univ. Padua, Italy). *Chir. Gastroenterol.* 8(2):131-136, 1974.

3972 STUDIES ON GASTRIC ACID SECRETION USING AN ISOLATED WHOLE MAMMALIAN STOMACH IN VITRO. *(E.)* Parsons, M. E. (Smith Kline French Lab., Ltd., Welwyn Garden City, England). *J. Physiol. (Lond.)* 247(1):35P-36P, 1975.

3973 IMMUNOREACTIVE GASTRIN VARIANTS IN CAT SERUM. *(E.)* Blair, E. L.; Grund, E. R.; Lund, P. K.; Reed, J. D.; Sanders, D. J. (Med. Sch., Univ. Newcastle upon Tyne, England). *J. Physiol. (Lond.)* 247 (1):33P-34P, 1975.

See also, 3887, 3988, 4376, 4439, 4488, 4495, 4496, 4500, 4504, 4505.

SECRETION AND METABOLISM

Pancreas

3974 PURIFICATION AND PROPERTIES OF SUBUNITS OF STEROL ESTER HYDROLASE FROM RAT PANCREAS. *(E.)* Calame, K. B.; Gallo, L.; Cheriathundam, E.; Vahouny, G. V.; Treadwell, C. R. (George Washington Univ. Med. Cent., Washington, D.C.). *Arch. Biochem. Biophys.* 168(1):57-65, 1975.

A procedure for the purification of subunits of sterol ester hydrolase and the results of studies concerning the nature of the interaction of enzyme subunits with cholic acid and its taurine conjugate were described. Subunits of sterol ester hydrolase from rat pancreas were solubilized by treatment of tissue ho-

mogenate with 0.5% digitonin. The crude, solubilized enzyme subunit was shown to have a molecular weight of approximately 70,000 by Sephadex G-200 gel filtration. The subunit was purified 500-fold by a combination of ammonium sulfate fractionation, hydroxylapatite, and gel-filtration chromatography. The subunits had been shown to aggregate in the presence of cholic acid or sodium taurocholate to a 400,000 molecular-weight form, which is the active enzyme. Three types of differential inactivation studies (thermal, guanidine hydrochloride, and pH) indicated significantly greater stability of the active enzyme when compared to the subunits. The data support the tentative conclusion that a conformational change or series of changes accompanies the binding of the bile salt to the enzyme subunits, which results in their aggregation and enzyme activity.

3975 IN VITRO STUDIES ON INTERACTION OF RAT PANCREATIC LIPASE AND COLIPASE WITH BILIARY LIPIDS. *(E.)* Lairon, D.; Nalbone, G.; Domingo, N.; Lafont, H.; Hauton, J.; Julien, R.; Rathelot, J.; Canioni, P.; Sarda, L. (Res. Group. Lipid Transp., INSERM, Marseilles, France). *Lipids* 10(4):262-265, 1975.

The interaction of lipase with biliary lipids was studied in the presence and absence of colipase. Rat pancreatic juice and bile were collected separately by cannulation, and rat pancreas was homogenized and centrifuged. The supernatant and the collected pancreatic juice were gel filtered, and the lipase and colipase fractions separated out. Rat bile (1.0 ml containing 3 mg/ml phospholipid) was mixed with either 0.1 ml of the lipase fraction (300 enzyme U), 1.2 ml of the colipase fraction (445 cofactor U); or 0.1 ml lipase and 1.0 ml colipase. The mixtures were stirred for 2 hr and filtered through a gel column. Lipase activity was determined at pH 9.0 and bile phospholipids were extracted with chloroform-methanol, and the phosphorus content estimated. When lipase was mixed with bile, the major part of the enzyme was eluted at 2.3 void volumes, and very little activity was associated with the phospholipids that emerged at 2.0 void volumes. A large portion of colipase was associated with these phospholipids, however. When both lipase and colipase were mixed with bile, the major enzyme activity was associated with the phospholipids in a high molecular weight complex including colipase. It is concluded that colipase is present in the complex formed by lipase with biliary or pancreatic lipids, that colipase is essential for the formation of the high molecular weight form of the enzyme, and for the attachment and function of lipase at interfaces.

3976 STUDIES ON DISPERSED PANCREATIC EXOCRINE CELLS. II. FUNCTIONAL CHARACTERISTICS OF SEPARATED CELLS. *(E.)* Amsterdam, A.; Jamieson, J. D. (Rockefeller Univ., New York, N.Y.). *Cell Biol.* 63(3):1057-1073, 1974.

Functional characteristics of guinea pig pancreatic exocrine cells isolated by a procedure described in a previous paper by the same authors were assessed by studying the ability of the cells to synthesize, transport, and concentrate exportable proteins into zymogen granules and to release the sorted products in response to secretogogues. The ability of isolated cells to incorporate labeled amino acids ([^{14}C] leucine) into secretory proteins was assessed biochemically and by quantitative electron microscope autoradiography. Incorporation remained linear for up to 4 hr incubation at levels equivalent to those of pancreatic slices and ranged from 1,700-2,000 nM leucine/mg DNA. Over 95% of the exocrine cells in the population were viable, and all appeared to be equally active in incorporating amino acids. The capacity of separated cells to transport, concentrate, and store exportable proteins was monitored by electron microscope autoradiography on populations pulse labeled with [^{3}H]leucine and chase incubated for periods up to 4 hr. The same overall pathway previously mapped in pancreatic slices was followed by secretory proteins in separated cells, although in quantitative studies a defect was noted in the rate of conversion of condensing vacuoles to zymogen granules. Secretogogue responsiveness was assessed by monitoring discharge of labeled secretory proteins or of amylase in response to carbamylcholine and cerulein. While the separated cells released secretory proteins linearly for up to 4 hr in response to both secretogogues, the net release was about 50% less than that previously noted for pancreatic slices and required a 10 times higher concentration of stimulant. The defect may represent alteration in receptors due to the protease used for dissociation. It is concluded that separated exocrine cells retain their ability to process secretory proteins stepwise and vectorially which is consistent with preservation of structural polarity and that these cells should prove useful and satisfactory for a number of studies not requiring prolonged incubation times. The system, however, exhibits a number of defects including those related to inefficient concentration of proteins in condensing vacuoles, suboptimal response to secretogogues, and a slow decay with time as evidenced by loss of DNA from the system.

3977 THE EFFECT OF ELECTRICAL VAGAL STIMULATION ON CANINE PANCREATIC EXOCRINE FUNCTION. *(E.)* Kaminski, D. L.; Ruwart, M. J.; Willman, V. L. (Dep. Surg., St. Louis Univ., Mo.). *Surgery* 77(4): 545-552, 1975.

The effect of electrical vagal stimulation on the canine pancreatic exocrine function was studied in conscious dogs by stimulating intact thoracic vagus nerves, the distal ends of cut vagus nerves in animals with intact gastric denervation, and the distal ends of cut vagus nerves in dogs whose stomachs had previously been selectively denervated. The dogs were fasted for 18 hr prior to each experiment, and during the experiments they received i.v. NaCl solution; gastric and pancreatic secretions were collected and measured every 15 min. Electrical stimulation of intact vagus nerves produced no significant change in the pancreatic output, the bicarbonate output, output of the secretin-stimulated pancreas, or protein output. Stimulation of both intact nerves did produce a significant increase in

protein concentration and an emetic response associated with increased mucus and acid production by the stomach, which dissipated during the stimulation period. Stimulation of the distal ends (efferent fibers) of the cut vagus nerves in dogs with intact gastric innervation significantly increased the volume and protein output of the pancreas and the acid output of the stomach. Stimulation of the distal ends of cut right and both vagus nerves in animals whose stomachs had been selectively denervated significantly increased the volume and protein output of the pancreas without increasing the output of acid by the stomach. The data indicate that the canine pancreas is innervated directly by vagal fibers, which when stimulated produce an increase in the protein (enzyme) output and the volume of secretion. Maintenance of the pancreatic response following denervation of the stomach suggests that the response is primarily the result of direct vagal innervation and is not produced by gastrin released from the antrum.

3978 EFFECT OF HYPOBARIC HYPOXIA ON PANCREATIC SECRETION STIMULATED BY FOOD AND BY INTRADUODENAL INFUSION OF HYDROCHLORIC ACID. *(Pol.)* Mroczka, J.; Sieppel-Maciejewska, K.; Bilski, R.; Konturek, S. (Acad. Med., Krakow, Poland). *Przegl. Lek.* 31(10):847-851, 1974.

Seven dogs with pancreatic fistulas were put into a baric chamber and pancreatic secretion stimulated in the first series of experiments by a mean meal (25 g/kg) and in the second series by intraduodenal infusion of 0.1 N hydrochloric acid (8 mEq/hr). The barometric pressure was decreased to approximately 1/2 atmosphere for 2 hr, and pancreatic juice was collected every 15 minutes. The volume and bicarbonate and protein outputs were determined and compared to controls. After the meat meal the volume of the pancreatic juice secreted during the first hr decreased 50% and during the second hr, 62% (22 and 13 ml, resp., as compared to 45 and 35 ml, resp. in controls). Bicarbonate output decreased 66% after 1 and 76% after 2 hr (1.95 and 1.05 mEq/hr, resp., as compared to 5.66 and 4.35 mEq/hr, resp. in controls). Protein contents decreased 30% after 2 hr (1315 mg and 895 mg resp. in the experimental group and 1343 mg and 1180 mg in controls). After the pressure returned to normal, the values of the measured parameters in the experimental group exceeded those in controls. When HCl was instilled into the duodenum the decreases were less pronounced than in the first series. The volume decreased 23%, bicarbonates 33%, and protein 33%. Lower values persisted in this series, even after pressure was increased to normal values. It is concluded that hypoxia connected with lowering of atmospheric pressure causes a marked inhibition of pancreatic secretion.

3979 ACTION OF SOME SYMPATHOMIMETIC AMINES UPON THE EXTERNAL SECRETION OF THE PANCREAS IN THE RAT. *(Fr.)* Roze, C.; Debray, C.; de La Tour, J.; Souchard, M.; Vaille, C. (Bichat Hosp., Paris, France). *J. Pharmacol. (Paris)* 5(2):155-172, 1974.

Male CF Wistar rats under urethane anesthesia with acute pancreatic fistula were studied for the effects of various substances on pancreatic secretion. Epinephrine and norepinephrine increased the output of pancreatic juice by a factor of 2.5 after 10 min of i.v. perfusion at concentrations of 0.13 μM/kg/min and 0.232 μM/kg/min, resp. Isoproterenol had the same effect, but it was more marked and occurred at lower concentrations. At a concentration of 0.05 μM/kg/min, pancreatic secretion rose to about 75% of the maximal secretory capacity induced by secretin. The logarithmic relation of dose/effect approximated a linear function. Propranolol (2 mg/kg i.v.) produced a moderate and prolonged decrease in the basal pancreatic output, and delayed and decreased the effect of isoproterenol administered after the propranolol. Phenoxybenzamine alone (2 mg/kg) had a marked and very prolonged stimulating effect on pancreatic output. Association of phenoxybenzamine and propranolol did not block the effect of isoproterenol more than did propranolol alone. Sparteine, which inhibits the hyperglycemia produced by the urethane anesthesia, was administered with isoproterenol to determine whether the urethane affected the apparent effect of isoproterenol. It was found that the effect of isoproterenol remained the same. The pancreatic output of bicarbonates is increased by isoproterenol stimulation. Total proteins and amylase output is either unchanged or slightly increased by the sympathomimetic amines, doubtless due to a washing out effect. Propranolol has a prolonged decreasing effect, and phenoxybenzamine has a prolonged increasing effect on the pancreatic output of proteins and amylase, which reflects variations in water output rather than in concentration of protein and amylase. The results suggest an α-adrenergic inhibitor and β-stimulant effect, which diverges from the classical picture of uniquely inhibitor sympathetic effects demonstrated in the dog, cat, and guinea pig. This evidence also diverges from the picture of stimulation of receptors associated with an increase in intracellular AMP_c concentration demonstrated *in vitro* for the rat, rabbit, cat, and mouse. It may be that in the anesthetized rat, AMP_c plays a role in hydrolytic secretion.

3980 METHADONE INHIBITION OF CENTRAL VAGAL PANCREATIC STIMULATION INDUCED BY 2-DEOXY-D-GLUCOSE. DESENSITIZATION BY REPEATED ADMINISTRATION. *(Fr.)* Vaille, C.; Roze, C.; de La Tour, J.; Souchard, M.; Debray, C. (Bichat Hosp., Paris, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 279(20):1641-1644, 1974.

A study was undertaken in Wistar CF rats to determine whether chronic administration of methadone had any effect on methadone-induced suppression of pancreatic stimulation produced by 2-deoxy-D-glucose (2DG). Rats were given methadone hydrochloride (5 mg/kg/day s.c., gradually increased to 7.5, 10, 12.5, and 15 mg/kg/day) over a 2 or 4 week period. After methadone treatment, rats were anesthetized with urethane and double biliary and pancreatic fistulas were established. After measurement of basal pancreatic secretion, 5 mg/kg methadone hydrochloride was injected s.c., followed, 5 min later, by 75 mg/kg i.v. of 2DG. Pancreatic secretion in these rats was compared with that in rats given 2DG alone or methadone plus 2DG without methadone pretreatment. In rats not pretreated with methadone,

injection of methadone followed by 2DG had no effect on the output of pancreatic juice or protein. In rats given methadone for 2 weeks, acute methadone injection suppressed and delayed the stimulation produced by 2DG. After 4 weeks of methadone pretreatment, acute methadone injection no longer blocked pancreatic stimulation induced by 2DG but increased it. Parallel changes occurred in total protein and amylase secretion. In rats pretreated with methadone, basal pancreatic secretion (both volume and total protein) was significantly increased after surgery, but returned to control values within a few hours. This increased basal secretion may be due either to catecholamine release induced by urethane anesthesia or by hyperfunction of vagal secretory neurons.

3981 EFFECT OF EXTRAGASTRIC AND TRUNCAL VAGOTOMY ON PANCREATIC SECRETION IN THE DOG. *(E.)* Debas, H. T.; Konturek, S. J.; Grossman, M. I. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Am. J. Physiol.* 228(4):1172-1177, 1975.

The effect of extragastric vagotomy on pancreatic secretion was examined in four mongrel dogs with chronic pancreatic and gastric fistulas and compared to the effect of subsequent transthoracic truncal vagotomy in the same animals. Dose-response studies of pancreatic bicarbonate and protein secretion were done with i.v. infusions of secretin, octapeptide of cholecystokinin, and 2-deoxyglucose. The pancreatic response to a meal of beef liver (15 g/kg) and intraduodenal perfusion of graded concentrations of HCl, sodium oleate, and tryptophan were also studied. Each stimulant was studied on two occasions in each of three stages: control; after extragastric vagotomy; and after transthoracic truncal vagotomy. During each test, the gastric fistula was kept open. Pancreatic juice was collected continuously and separated into 15-min samples. The responses to secretin, octapeptide of cholecystokinin, and to duodenal HCl perfusion were either unaltered or only slightly decreased by either extragastric or truncal vagotomy. Basal pancreatic secretion and the responses to duodenal perfusion of oleate and tryptophan were markedly depressed by extragastric vagotomy. Extragastric vagotomy also reduced pancreatic responses to a meal and to 2-deoxyglucose. Subsequent truncal vagotomy caused still further reduction, possible by depressing antral gastrin release. These findings indicate that tonic vagal activity contributes to basal pancreatic secretion but has little effect on the response of the pancreas to secretin or cholecystokinin, or on the release of secretin from the intestine. It is suggested that the decreased pancreatic response to intestinally perfused oleate and tryptophan seen after extragastric vagotomy could be caused either by interruption of reflex paths between gut and pancreas or by interference with cholecystokinin. It is concluded that any advantage that selective vagotomy may have over truncal vagotomy in the treatment of duodenal ulcer probably cannot be attributed solely to an effect on the pancreas.

3982 SECRETORY AND METABOLIC EFFECTS OF ALCOHOL ON THE PANCREAS. *(E.)* Webster, III, P. D. (VA Hosp., Augusta, Ga.). *Ann. NY Acad. Sci.* 252: 183-186, 1975.

3983 EFFECT OF ANTIBIOTICS ON EXOCRINE PANCREATIC FUNCTION. *(Ger.)* Fleischer, K. (Med. Clin., Univ. Wurzburg, Germany). *Ber. Phys. Med. Ges. Wurzburg* 82:45-51, 1974.

3984 STUDY OF EXOCRINE SECRETION FROM DUODENO-PANCREATIC HOMOTRANSPLANTS IN THE DOG. *(Fr.)* Gautier-Benoit, C.; Pinchon-Marsy, S.; Pinchon, J.; Chambon, J.; Daboust, M.; Wattine, M.; Hardy, E.; Duwat, R. (Hosp. Cent., Lens, France). *Lille Chir.* 29(3):77-79, 1974.

3985 RELATIONS BETWEEN PANCREATIC AND BILE SECRETION: EXPERIMENTS ON DOGS. *(Ger.)* Nowak, W.; Sill, U.; Lippert, H.; Reding, R. (Surg. Clin., Ernst Moritz Arndt Univ., Greifswald, Germany). *Zentralbl. Chir.* 99(41):1288-1291, 1974.

3986 INTRADUODENAL CALCIUM STIMULATES THE LIBERATION OF CHOLECYSTOKININ-PANCREOZYMIN (CCK-PZ) IN MAN. *(Ger.)* Holtermuller, K. H.; McCall, J. T.; Malagelada, J. R.; Go, V. W. L. (Med. Clin., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:536-537, 1974.

3987 INFLUENCE OF DIMINISHED MOTOR ACTIVITY ON THE ENZYME EXCRETORY FUNCTION OF THE PANCREAS AND THE EXTERNAL SECRETORY FUNCTION OF THE LIVER OF RATS. *(Rus.)* Smirnov, K. V.; Goland, L. G.; Medkova, I. L. (no affil.). *Kosm. Biol. Aviakosm. Med.* 8(3):22-26, 1974.

3988 CHANGES IN SERUM GASTRIN LEVELS AFTER INTRADUODENAL ADMINISTRATION OF BILE. *(Ger.)* Londong, W.; Fruhauf, S.; Forell, M. M. (2nd Med. Clin., Univ. Munich, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:516-518, 1974.

3989 PERIODIC CHANGES IN PANCREATIC ENZYME SECRETION DURING FASTING. *(Rus.)* Shligin, G. K.; Laktaeva, T. V. (Inst. Nutr., Moscow, USSR). *Fiziol. Zh. SSSR* 61(4):628-635, 1975.

3990 THE MECHANISM OF ACTION OF PARENTERALLY ADMINISTERED PROTEIN HYDROLYSATES ON EXOCRINE PANCREAS. *(Rus.)* Sisoiev, Iu. A.; Shlygin, G. K. (Inst. Nutr., Moscow, USSR). *Fiziol. Zh. SSSR* 61(3):423-427, 1975.

3991 THE EFFECT OF ANTIBACTERIAL PREPARATIONS ON THE ACTIVITIES OF PROTEINASES IN PANCREATIC JUICE AND ON THE CONTENT OF SOME ENZYMES IN GASTROINTESTINAL ORGANS. *(Rus.)* Storozhuk, P. G.; Plakhotiniukova, V. V.: Tsynkalovskii, I. B. (Kuban Med. Inst., Krasnodar, USSR). *Vopr. Med. Khim.* 20(4):343-349, 1974.

3992 ACTIVITIES OF ELASTASE AND PROELASTASE IN RAT PANCREAS IN EXPERIMENTAL PANCREATITIS. *(Rus.)* Stefanov, A. V.; Levitskii, A. P. (Res. Inst. Stomatol., Odessa, USSR). *Vopr. Med. Khim.* 20(6): 634-637, 1974.

3993 DNA REPAIR SYNTHESIS FOLLOWING EXPOSURE OF GUINEA-PIG PANCREATIC SLICES TO METHYL-N-NITROSOURETHANE IN VITRO. *(E.)* Hasumi, K.; Iqbal, Z. M.; Alarif, A.; Epstein, S. S. (Case West. Reserve Univ. Sch. Med., Cleveland, Ohio). *Experientia* 31(4): 467, 1975.

See also, 3882, 3952, 4177.

SECRETION AND METABOLISM

Hepatobiliary Tract

3994 EFFECT OF PHENOBARBITAL, SPIRONOLACTONE AND PREGNENOLONE-16α-CARBONITRILE ON BILE FORMATION IN THE RAT. *(E.)* Von Bergmann, K.; Schwarz, H. P.; Paumgartner, G. (Inst. Clin. Pharmacol., Univ. Bern, Switzerland). *Naunyn. Schmiedeberg's Arch. Pharmacol.* 287(1):33-45, 1975.

The effects of phenobarbital, spironolactone, and pregnenolone-16 α-carbonitrile on bile flow and bile lipid secretion were studied in adult male Sprague-Dawley rats. Phenobarbital sodium (8 mg/100 g, once daily), spironolactone (10 mg/100 g, twice daily), pregnenolone-16α-carbonitrile (3.5 mg/100 g, twice daily), saline, or gum arabic in water were administered by stomach tube for 4 days. Twenty-four hr after the last infusion, the bile duct was cannulated and the bile collected over three 10-min periods. In a second study, rats pretreated as above were infused with sodium taurocholate and the effect on the bile determined as above. Phenobarbital, spironolactone, and pregnenolone-16α-carbonitrile increased bile flow but did not alter bile salt excretion. During bile salt infusion, phenobarbital decreased the secretion of cholesterol and phospholipid to 39% and 49% of control values, resp.; however, spironolactone and pregnenolone-16α-carbonitrile more than doubled cholesterol excretion without influencing the output of phospholipid. As a result, phenobarbitol decreased the degree of cholesterol saturation while spironolactone and pregnenolone-16α-carbonitrile increased it. The findings indicate that different types of enzyme inducers may differ in their effects on biliary lipid secretion. Thus, depending on its influence on the cholesterol saturation of the bile, an inducer could contribute to or prevent the formation of cholesterol gallstones.

3995 HORMONAL CONTROL OF THE POTASSIUM LEVEL IN ISOLATED RAT LIVER CELLS. *(E.)* Barnabei, O.; Leghissa, G.; Tomasi, V. (Lab. Comp. Physiol., Univ. Bologna, Italy). *Biochim. Biophys. Acta* 362 (2):316-325, 1975.

Liver cells isolated from nonfasted female Wistar rats were incubated in media containing epinephrine, theophylline, insulin, ouabain, and/or glucagon, and the effects on potassium uptake and on the cyclic AMP levels of the cells were monitored. Liver cells isolated by the method of Berry and Friend (1969) contained about 48 μEq K+/g. Although 44% of the potassium was lost when the cells were kept at 4 C for 5 hr, the initial ion levels were restored by incubation at 37 C. Ouabin, epinephrine, and epinephrine plus theophylline inhibited the uptake of potassium by these cells; insulin partially counteracted the inhibiting effect of epinephrine, but had no effect on the basal potassium levels. Liver cells isolated by the procedure of Seglen (1972) were able to synthesize glycogen from glycose and contained a much higher level of potassium (102.2 μEq/g). Again, epinephrine and theophylline inhibited the uptake of K+, as did monobutyryl cyclic AMP and ouabain. Epinephrine and theophylline, alone and in combination, also increased the cellular levels of cyclic AMP; both epinephrine and glucagon produced, respectively, a 2- and 4-fold increase in cyclic AMP after only 2 min of incubation at 37 C. Ouabain and insulin had no effects on the cyclic AMP levels. The results suggest that the epinephrine-stimulated efflux of K+ in isolated liver cells may be due to an inhibition of the activity transport of potassium; this effect is probably mediated by cyclic AMP.

3996 EFFECT OF DIPHENYLHYDANTOIN ON THE HEPATIC MICROSOMAL ETHANOL OXIDIZING SYSTEM IN THE RAT. *(E.)* Andreasen, P. B.; Bremmelgaard, A. (Rigshosp., Copenhagen, Denmark). *Pharmacology* 12(4-5): 237-243, 1974.

The effect of diphenylhydantoin (DPH) on the hepatic microsomal ethanol-oxidizing system was investigated in female Wistar rats. The animals received 5-10 or 33-37 daily i.p. injections of DPH (75 mg/kg) or isotonic saline, after which the liver was removed and the microsomal protein and cytochrome P-450 content and lactate dehydrogenase and ethanol-oxidizing activities were determined. In the rats treated with DPH for 5-10 days, the microsomal lactate dehydrogenase activity was significantly elevated over control values; all other parameters were within normal limits. In the rats treated for 33-37 days, the liver weight, lactate dehydrogenase activity, cytochrome

P-450 content, and microsomal ethanol-oxidizing activity were all significantly elevated over control values. The hepatic cytochrome P-450 content and microsomal ethanol-oxidizing activity were significantly correlated, as were the hepatic lactate dehydrogenase activity and the microsomal ethanol-oxidizing activity. The results suggest that the interaction between ethanol and DPH metabolism may be mediated *in vivo* at the microsomal level.

3997 OXIDATION OF ETHANOL BY HEPATIC MICROSOMES OF ACATALASEMIC MICE. *(E.)* Lieber, C. S.; DeCarli, L. M. (Bronx VA Hosp., N.Y.). *Biochem. Biophys. Res. Commun.* 60(4):1187-1192, 1974.

Acetaldehyde production was used to assess ethanol oxidation by the liver microsomes of acatalasemic mice. The hepatic microsomes of Cs^b acatalasemic and Cs^a control mice were incubated *in vitro* with ethanol (50 mM) and a NADPH-generating system, and the activity of the microsomal ethanol-oxidizing system (MEOS) was measured. When subjected to heat inactivation, the liver microsomes of the actalasemic mice displayed decreased catalytic activity, but the NADPH-dependent MEOS remained unaffected. Even without heat inactivation, the NADPH-dependent metabolism of ethanol was much more active than the H_2O_2-mediated system, while the microsomes of the control mice showed equal rates of H_2O_2- and NADPH-dependent ethanol oxidation. The addition of catalase to the Cs^b liver microsomes *in vitro* markedly increased the capacity of the microsomes to oxidize ethanol in the presence of an H_2O_2-generating system and abolished this difference between the 2 strains of mice. In the presence of the catalase inhibitor azide, control Cs^a microsomes behaved similarly to the microsomes of the Cs^b animals. The selective persistence in the Cs^b mice of NADPH-dependent ethanol oxidation contrasting with the reduction in the H_2O_2-mediated metabolism of ethanol supports the existence of a catalase-independent ethanol-oxidizing system.

3998 PLASMA AND HEPATIC BINDING OF INDOCYANINE GREEN IN GUINEA PIGS OF DIFFERENT AGES. *(E.)* Hwang, S. W. (Natl. Inst. Environ. Health Sci., Research Triangle Park, N.C.). *Am. J. Physiol.* 228 (3):718-724, 1975.

The binding of indocyanine green (ICG) to the plasma and bile proteins of guinea pigs of varying ages was studied as was the uptake and efflux of ICG from liver slices. Protein binding was studied by a gel-filtration method in which plasma or bile samples from guinea pigs ranging in age from 1 day to adult were eluted with ICG in Sephadex columns. An ultracentrifugation method was used to study ICG binding to liver tissue, and the uptake of ICG by and efflux of ICG from liver slices was studied by placing fresh liver slices in beakers containing Krebs-Ringer-phosphate medium. The ICG in the plasma was almost totally bound to the plasma protein and there was no significant difference in binding between newborn and adult animals. The binding of ICG to biliary protein was also similar in all age groups. However, particulate fractions of liver from adult guinea pigs showed a higher ICG binding capacity than those from newborn animals at all ICG concentrations measured. The uptake of ICG by liver slices involved mostly active processes. The slice-to-medium (S/M) ratio of ICG was lowest in the liver preparations from the newborn animals, but the S/M ratio for adult animals was lower than that for 10-day-old animals. Liver slices from adult guinea pigs showed the fastest ICG efflux rates, the slowest rates being observed with the slices from the newborn animals. The results suggest that the degree of ICG binding to plasma and biliary proteins is not significantly different in newborn and adult guinea pigs. The incomplete development of ICG uptake and release mechanisms in the liver of the newborns may at least partially account for the lower biliary excretory capacity for ICG in the neonate.

3999 PHOSPHOLIPIDS AS CINCHOCAIN ACTION SITE DURING INHIBITION OF LIPOPEROXIDATIVE REACTIONS IN RAT LIVER MICROSOMES. *(E.)* Mishin, V.; Tsyrlov, I.; Gromova, O.; Lyakhovich, V. (State Inst. Med. Novosibirsk, USSR). *Hoppe Seyler's Z. Physiol. Chem.* 355(5):626-632, 1974.

The mechanism and site of action involved in the dibucaine hydrochloride-induced inhibition of microsomal lipid peroxidation was studied. Liver microsomes from male Wistar rats were incubated with dibucaine hydrochloride and NADPH- and ascorbate-dependent lipid peroxidation; the peroxidation activity of cytochrome P-450, the activity of NADPH-cytochrome reductase, and the binding of cinchocain with cytochrome P-450 were measured. Dibucaine hydrochloride inhibited oxygen consumption during NADPH- and ascorbate-dependent lipid peroxidation, but did not affect the cytochrome P-450 catalyzed decomposition of linoleic acid hydroperoxide. Dibucaine hydrochloride also did not hinder the process of microsomal electron transport from NADPH to cytochrome P-450. Metyrapone, a classical type II substrate, competitively interferred with the binding of dibucaine hydrochloride to cytochrome P-450, and phosphatidylethanolamine markedly weakened the inhibiting effect of dibucaine hydrochloride on microsomal lipid peroxidation. In addition, dibucaine hydrochloride considerably raised liver microsome hydrophobicity, increasing the quantum yield of 8-aniline-1-naphthalene sulfonate fluorescence in the lipid phase. The results suggest that an increased phospholipid hydrophobicity and a resultant decreased accessibility of the phospholipids for peroxidative reactions is responsible for the dibucaine hydrochloride-induced inhibition of microsomal lipid peroxidation.

4000 ACUTE CHOLESTASIS INDUCED BY LITHOCHOLIC ACID IN THE RAT. A FREEZE-FRACTURE REPLICA AND THIN SECTION STUDY. *(E.)* Miyai, K.; Mayr, W. W.; Richardson, A. L. (Sch. Med., Univ. California San Diego, La Jolla). *Lab. Invest.* 32(4):527-535, 1975.

Ultrastructural changes in lithocholic acid-induced cholestasis were studied by freeze-fraction techniques in female Wistar rats with bile fistulas. Sodium

lithocholate was continuously infused i.v. (0.1 or 0.2 μM/min/100 g) for up to 4 hr. The higher dose induced complete cholestasis within 2-3 hr; the lower dose reduced the biliary output to less than 10% of the preinfusion level by the 3rd hr. Ultrastructural changes, primarily localized to the bile canaliculi and the pericanalicular region, were seen 30 min after the onset of bile acid infusion. Dilation of the bile canaliculi, loss of canalicular microvilli, prominence of the pericanalicular ectoplasm, and a characteristic lamellar transformation of the canalicular membrane developed, which become more prominent and widespread with time. The freeze-fracture replica study revealed that the canalicular microvilli became transformed through widening and flattening into multilamellar foldings. Intramembranous granules of the canalicular membrane appeared to have become redistrubuted, being few or absent in the transformed regions. In addition, a sharply angulated, crystalline material was seen in occasional bile canaliculi. This material appeared as a negative image in thin sections, indicating its solubility in organic solvents which were used for dehydration. With the lower dose of lithocholic acid, subcellular changes were similar to, but less severe and widespread than, those induced by the high dose. It is suggested that the hepatic ultrastructural changes which accompany lithocholic acid-induced acute cholestasis are attributable to the accumulation of this compound in the bile canaliculus and its vicinity. Lithocholic acid presumably causes an asymmetric perturbation in the molecular organization of the canalicular membrane which results in ultrastructural alterations and failure of fluid transport. In addition, precipitates of lithocholic acid appear to form in the bile canaliculi and may contribute to cholestasis.

4001 AN ETIOLOGIC BASIS FOR FATTY LIVER AFTER JEJUNOILEAL BYPASS. *(E.)* Hollenbeck, J. I.; O'Leary, J. P.; Maher, J. W.; Woodward, E. R. (Coll. Med., Univ. Florida, Gainesville). *J. Surg. Res.* 18(2):83-89, 1975.

An investigation undertaken in an attempt to prevent liver failure and fatty metamorphosis after jejunoileal bypass in experimental animals is described. An obesity bypass procedure was performed on eight healthy mongrel dogs. The intestine was divided 24 in from the pylorus and anastomosed end-to-side to the terminal ileum 12 in from the ileocecal valve. The blind end of the bypassed intestine was closed and a liver wedge biopsy performed. At subsequent monthly intervals the animals were operated upon, wedge biopsies were obtained, and aerobic and anaerobic cultures of bacterial flora were taken 12 in from the proximal end of the excluded limb. Four of the dogs received doxycycline hyclate, 100 mg/day p.o., while the other four received no antibiotics. All of the dogs survived the original procedure and experienced diarrhea as a sequelae of the operation; the diarrhea subsided within 10 days of the operation. Bacteriologic studies of the cultures taken from the defunctionalized intestine revealed *Bacteroides fragilis, Bacteroides species,* or both in all of the animals in the control group while none of the animals treated with doxycycline hyclate grew *Bacteroides*. It is suggested that *Bacteroides* organisms within the bypassed segment produce a hepatotoxic endotoxin. It was proposed that after obesity bypass, *Bacteroides*-specific antibiotics given p.o. may prevent post-bypass liver failure.

4002 BIOCHEMISTRY OF PEROXYSOME IN LIVER CELLS. *(Ger.)* Sies, H. (Inst. Physiol. Chem. Phys. Biochem., Univ. Munich, Germany). *Angew. Chem.* 86(22):789-824, 1974.

Biochemical studies on hepatocellular peroxysome are reviewed. The principal enzyme of peroxysome is catalase which is able to metabolize hydrogen peroxide both catalytically and peroxydatically. Catalase in peroxysome is associated with hydrogen peroxide-supplying oxidases. An active intermediate catalase-Fe^{3+}-H_2O_2 (complex I) is responsible for the peroxysomal H_2O_2 metabolism. Organ photometric investigations of intact, hemoglobin-free perfused rat liver for the selective measurement of Complex I is suitable for the dynamic study of the H_2O_2 metabolism taking place in the nanomol range. The frequency (quotient of rate of H_2O_2 formation and catalase heme concentration) can be increased to about 100/min by intracellular stimulation of the H_2O_2 production by glycolate and urate, against less than 10/min as measured in steady state. The predominantly peroxidatic oxidation of hydrogen donors, such as methanol and ethanol, at low frequencies at the expense of catalatic oxidation is of significance for normal and pathological metabolism. The catalatic oxidation of ethanol and methanol is stimulated by intensified H_2O_2 formation. Disturbances in hepatic metabolism in alcoholic states may be influenced by the administration of pyrazole derivatives with simultaneous stimulation of the peroxidatic metabolism of alcohol.

4003 ENZYME PROFILES OF MAMMALIAN BILE. *(E.)* Holdsworth, G.; Coleman, R. (Dep. Biochem., Univ. Birmingham, England). *Biochim. Biophys. Acta* 389(1):47-50, 1975.

The relations of the specific activities of bile enzymes to those in the hepatocytic plasma membrane was investigated. Bile was obtained from ox, sheep, pig, and rat, and liver homogenates were also prepared from these animals. Bile samples were also obtained from human patients. The following enzymes were assayed in both the bile samples and their corresponding liver homogenates: 5'-nucleotidase, alkaline phosphodiesterase I, L-leucyl-β-napthylamidase, Mg^{++}-ATPase, acid phosphatase, aryl sulfatase, succinate dehydrogenase, aryl esterase, LDH, and alcohol dehydrogenase. There were significant amounts of alkaline phosphodiesterase I, and smaller amounts of L-leucyl-β-naphthylamidase, 5'-nucleotidase and alkaline phosphatase (the four plasma membrane-bound enzymes) in the bile of each species. The intracellular enzymes (acid phosphatase, succinate dehydrogenase, LDH, and alcohol dehydrogenase) were completely absent from the bile. Polypeptide profiles of the bile samples as determined by electrophoresis showed a small number of

bands when compared with those of the corresponding liver homogenates. It is concluded that preferential release of plasma membrane protein components is a consistent feature of bile salt secretion.

4004 INDUCTION OF HEPATIC ALKALINE PHOSPHATASE BY A SUBSTANCE IN BILE. *(E.)* Jerksy, J. (Dep. Surg., Univ. Witwatersrand, South Africa). *S. Afr. J. Surg.* 12(4):255-257, 1974.

The effects of bile infusion on the production of serum alkaline phosphatase was studied in normal baboons. Bile was aspirated from the gallbladders of normal baboons and fractionated into 5 fractions, and 50 ml of each fraction was infused into 4 additional baboons. Infusion of whole bile was followed by a sharp rise in the serum alkaline phosphatase levels. Similar increases were noted after the infusion of the low-molecular weight and water-soluble fraction, but not after infusion of the high-molecular weight and ethanol-chloroform-soluble fractions. The whole bile and all fractions except the low-molecular weight fraction contained alkaline phosphatase activity. That a fraction which caused a rise contained no alkaline phosphatase supported the conclusion that the effect on enzyme levels was due to a substance in bile. Since the filter used for bile fractionation retains molecules with a molecular weight greater than 10,000 and as the filtrate contained the active ingredient, its molecular weight must be less than 10,000. Similarly, the stimulating substance must be water soluble. In hepato-biliary diseases this agent, which is produced in the liver and excreted in the bile, may be diffused into the circulation. From there, it would reach the liver where it could induce increased alkaline phosphatase production by all liver cells, whether obstructed or not. Such a mechanism would explain why alkaline phosphatase is often elevated in conditions where the serum bilirubin is normal.

4005 THE ENZYMATIC FORMATION OF BILIRUBIN. *(E.)* Schmid, R.; McDonagh, A. F. (Dep. Med., Univ. California, San Francisco). *Ann. NY Acad. Sci.* 244:533-552, 1975.

The enzymatic formation of bilirubin *in vitro* and *in vivo* is reviewed. The biological and chemical systems that convert heme to bile pigment *in vitro* are discussed and three biological systems are described: 1) a light-mitochondrial fraction from the dog placenta; 2) cultured chicken macrophages; and 3) a microsomal enzyme system, heme oxygenase, obtained from spleen, liver, bone marrow, and other tissues. Results of experiments in these systems suggest that the third is responsible for the catabolism of heme *in vivo* and plays a major role in the conversion of hemoglobulin to bilirubin. The following sequence of reactions is probably involved in the formation of bilirubin IX α *in vivo*: *meso*-hydroxylation of heme at the α-bridge; autoxidation of the resulting α-hydroxyheme to carbon monoxide and a biliverdin-iron complex; hydrolysis of the iron complex to biliverdin IXα ; and finally, enzymatic reduction of biliverdin IXα to bilirubin IXα by biliverdin reductase. The mechanism of the initial event, the critical step, is still incompletely understood. *In vitro* studies suggest that heme, bound to a protein, utilizes NADPH and the microsomal electron transport system to reduce oxygen and generate a reactive radical which attacks the heme at the α-bridge to form α-hydroxyheme. In this model, therefore, bound heme catalyzed its own hydroxylation with a binding protein serving as a substrate-specific enzyme. Chemical evidence suggests that the structure of this heme-binding protein is such that the heme is bound in a specific orientation within a crevice and with a strong ligand coordinated to the iron. It is uncertain whether the heme-binding protein, which is equated with the microsomal heme oxygenase discussed under *in vitro* biological systems, is a unique protein specific to the endoplasmic reticulum, or whether other membrane-bound proteins can function in the same capacity.

4006 A MICROPUNCTURE STUDY OF RENAL SALT AND WATER RETENTION IN CHRONIC BILE DUCT OBSTRUCTION. *(E.)* Bank, N.; Aynedjian, H. S. (Montefiore Hosp. Med. Cent., Bronx, N.Y.). *J. Clin. Invest.* 55(5):994-1002, 1975.

The mechanism of sodium retention by the kidney of white male Sprague-Dawley rats with common bile duct ligation was studied with micropuncture techniques. Before surgery, an average of 93.2% of the ingested sodium was recovered in the urine over a 3-day observation period. Only 85.6% of the ingested sodium was recovered in the urine 10-14 days after bile duct ligation. This difference of 7.6% represents a positive sodium balance of approximately 142 μEq/day. Measurements of renal blood flow and glomerular filtration rate yielded values that were not different from those in normal control animals. Single nephron filtration rate of surface nephrons was the same in the experimental rats as in the controls. Sodium reabsorption, however, was markedly increased in the proximal convoluted tubule, as well as in segments beyond the proximal convolutions. Single nephron filtration fraction, calculated from measurements of efferent arteriolar and arterial hematocrits, was significantly elevated in the cortical nephrons, even though whole kidney filtration fraction was the same as in normal rats. The calculated protein concentration of cortical peritubular blood was higher in the bile duct-ligated rats than in the normal controls. These observations are consistent with the view that sodium retention is the result of enhanced reabsorption primarily by cortical nephrons. It is suggested that the enhanced reabsorption can be accounted for by relative cortical ischemia due to efferent arteriolar vasoconstriction with the consequent elevation of peritubular colloid oncotic pressure.

4007 BIOSYNTHESIS OF UDP-GLUCOSE BY RAT LIVER MICROSOMES. *(Fr.)* Berthillier, G.; Got, R. (Membr. Biochem. Lab., CNRS, Villeurbann, France). *Biochim. Biophys. Acta* 362(2):390-402, 1974.

A microsome fraction was obtained from Wistar rat livers by homogenization and centrifugation, and the supernatant was used to assay the concentration of soluble enzymes. Intermediary metabolites in the biosynthesis of uridine diphosphate glucose (UDP-glucose) were assayed by incubation of a suspension of microsomes in the presence of ^{14}C-glucose followed by analysis by column and thin layer chromatography; and by incubation of microsomes in the presence of glucose 1-phosphate, glucose-6-phosphate, or glucose followed by analysis of the concentration of synthesized UDP-glucose. After 15 min incubation of microsomes in the presence of ^{14}C-glucose, the quantities of glucose-6-phosphate, glucose-1-phosphate, and UDP-glucose formed were 0.96 m, 0.27 m, and 0.06 m, resp. For incubation of microsomes with any of the three precursors, equivalent quantities of UDP-glucose were formed. These results imply that phosphoglucomutase and UTP:D-glucose-1-phosphate uridyl transferase are present in the microsome membranes. The K_m of microsome membrane phosphoglucomutase was found to be 0.26 x 10^{-3} mM for glucose-1,6-diphosphate and 80 x 10^{-3} mM for glucose-1-phosphate at optimum pH and temperature. For soluble phosphoglucomutase, the respective K_m's were 0.4 and 40 x 10^{-3} mM. The K_m of microsome membrane UTP-D-glucose-1-phosphate uridyl transferase was found to be 220 x 10^{-3} mM for UTP and 166 x 10^{-3} mM for glucose-1-phosphate. For soluble UTP-D-glucose-1-phosphate uridyl transferase, the K_m's of UTP were 150 and 200 in and not in the presence of Triton X-100, resp., and for soluble UTP-D-glucose-1-phosphate uridyl transferase, the K_m of glucose-1-phosphate was 100 x 10^{-3} mM. It seems likely that there exists a double pathway, one soluble and one in the membrane, for UDP-glucose synthesis. The absence of accumulation of intermediary metabolites provides further evidence for an organized system of UDP-glucose synthesis by the membrane of rat liver microsomes.

4008 ROLE OF THE LIVER IN COAGULATION AND FIBRINOLYSIS: PATHOPHYSIOLOGICAL AND EXPERIMENTAL ASPECTS OF THE ISOLATED LIVER. *(Fr.)* Losito, R. (Dep. Med., Univ. Sherbrooke, Canada). *Union Med. Can.* 103(12):2107-2110, 1974.

Isolated and perfused rat liver was used to study the role of vitamin K, oral anticoagulants, and fibrinolysis in hepatic hemostasis. ^{14}C-Menadione (vitamin K_3) was readily metabolized, and approximately 3/4 of the dose was excreted by both normal and hepatectomized rats. Normal and hepatectomized rats, resp., excreted only 10% and 0.5% of an injected dose of vitamin K_1. This shows that the liver is essential to the metabolism of vitamin K, but not of menadione. It also explains the superiority of vitamin K as an antidote for an overdose of oral anticoagulant. Experiments with ^{14}C-warfarin in the isolated and perfused liver show that it is also excreted more rapidly than vitamin K, although the warfarin activity in the plasma remains essentially intact. The radioactivity in the liver introduced by both warfarin and vitamin K is associated with the microsomes and the supernatant material. Experiments with the fibrinolysin streptokinase show that a certain quantity of plasmin was produced in the perfusate, and different antiplasmins could neither neutralize it nor eliminate it from the liver. Fibrinolytic activity existed in the bile during the entire time of perfusion with streptokinase.

4009 THE EFFECT OF BILE ACID SYNTHESIS ON CHOLESTEROL SECRETION INTO THE BILE. *(E.)* Strasberg, S. M.; Ilson, R. G. (Toronto West. Hosp., Canada). *Ann. Surg.* 181(4):458-465, 1975.

Biliary cholesterol and bile acid secretion were studied in three female Rhesus monkeys during the period of increasing bile acid secretion and synthesis occurring 6-10 hr after interruption of the enterohepatic bile acid circulation by cannulation of the common bile duct. The bile acid pool was prelabeled by injection of ^{14}C-cholic acid 24 hr prior to start of the experiment. Bile acid synthesis and phospholipid secretion were increased 3- and 1.5-fold, resp., while cholesterol secretion was decreased 50-60% 40 hr after interruption of the hepatic circulation. Phenobarbital (5 mg/kg/24 hr i.v.) had no effect on the phospholipid secretion rate but did enhance cholesterol secretion and bile acid synthesis. These results suggest that bile acid synthesis influences cholesterol secretion into bile.

4010 ETHANOL - ANTIBIOTIC INTERACTIONS AT HEPATIC LEVEL. *(E.)* Grassi, G. G.; Grassi, C. (Inst. Chemother., Univ. Pavia, Italy). *Int. J. Clin. Pharmacol.* 11(3):216-225, 1975.

The effects of acute and chronic ethanol intoxication on blood levels of several antibiotics were studied in fasted male Wistar rats. Acute ethanol intoxication (2 mg/kg 50% ethanol i.v. or 5 mg/kg p.o.) had no effect on blood levels of isoniazid, rifampicin, streptomycin, kanamycin or gentamycin. Although chronic intoxication (5 mg/kg 50% ethanol 5-30 days by gastric intubation) did not alter blood aminoglycoside levels, rifamypicin levels were decreased by 20-50% and isoniazid concentrations were increased 40-80%. Rifampicin impaired hepatic BSP elimination in both acute and chronic experiments with or without alcohol intoxication. Isoniazid affected BSP elimination only in chronically intoxicated and control animals. These studies indicate that ethanol can affect degradation of certain antibiotics and suggest that antibiotic dosage may need modification in alcoholic patients.

4011 ALTERATION BY PHENOBARBITAL AND 3-METHYLCHOLANTHRENE OF FUNCTIONAL AND STRUCTURAL CHANGES IN RAT LIVER DUE TO CARBON TETRACHLORIDE INHALATION. *(E.)* Shah, H. C.; Carlson, G. P. (Coll. Pharm., Univ. Rhode Island, Kingston). *J. Pharmacol. Exp. Ther.* 193(1):281-292, 1975.

The effect of carbon tetrachloride (CCl_4) inhalation

(1100 ppm, 30 min) on hepatocyte polyribosome profiles, ^{14}C-leucine incorporation into liver protein and hepatocyte ultrastructure were studied in male Charles River rats pretreated with phenobarbital (50 mg/kg day for 4 days i.p.) or 3-methylcholanthrene (40 mg/kg/day for 2 days i.p.). The liver 9000 x g supernatant polyribosomal pattern was completely disrupted following CCl_4 inhalation in phenobarbital or 3-methyl-cholanthrene pretreated rats and returned to normal after 48 and 24 hr, resp. Incorporation of label into 9000 x g supernatant proteins was inhibited by 58%, 18% and 2% after 0, 24 and 48 hr in phenobarbital-treated rats and by 45% and 13% after 0 and 6 hr in three methycholanthrene-treated animals. The centrilobular hepatocytes of phenobarbital-pretreated rats exposed to CCl_4 showed dilation and vesiculation of cisternea of the rough endoplasmic reticulum as well as dilatation of the nuclear membrane space. Partial recovery occurred within 24 hr and complete recovery was seen 48 hr after exposure to CCl_4. No changes in the nuclear membrane or rough endoplasmic reticulum were observed in CCl_4 3-methylcholanthrene-treated animals. These results are explained by hypothesizing two activation sites for CCl_4 on the reduced NAD-cytochrome P-450 electron transport chain of the liver microsomes.

4012 VITAMIN B_6 METABOLISM IN CHRONIC ALCOHOL ABUSE. THE EFFECT OF ETHANOL OXIDATION ON HEPATIC PYRIDOXAL 5'-PHOSPHATE METABOLISM. *(E.)* Veitch, R. L.; Lumeng, L.; Li, T. K. (Indiana Univ. Sch. Med., Indianapolis). *J. Clin. Invest.* 55(5): 1026-1032, 1975.

The effect of chronic alcohol ingestion on hepatic vitamin B_6 metabolism was studied *in vivo* and *in vitro*. Male Sprague-Dawley rats fed a diet containing ethanol (36% of total calories) and pyridoxine (0.725 µg/ml) for 52 days had a significantly lower ($p < 0.02$) liver pyridoxal phosphate level (7.6 µg/200 mg protein) than animals fed a similar diet containing dextrin maltose instead of ethanol (8.5 µg/200 mg). Liver pyridoxal phosphate levels were also lower in rats fed an ethanol diet for 42 days without vitamin B_6 supplementation (3.0 µg/200 mg protein) than in controls devoid of B_6 supplementation (4.7 µg/200 mg). Perfusion of livers from B_6-sufficient rats with 18 mM ethanol for 3 hr lowered pyridoxal phosphate content by 3.8 µg/200 mg protein ($p<0.02$) compared to increase of 1.0 µg/200 mg in controls. Whereas release of preformed pyridoxal phosphate from perfused B_6-sufficient livers was linear for 3 hr, none was released from perfused B_6-deficient organs. Addition of pyridoxine to the perfusion medium of deficient livers resulted in a striking increase in both pyridoxal phosphate content and its release which could be partially inhibited by 18 mM ethanol. 4-Methylpyrazole (0.24 mM), a competitive inhibitor of liver alcohol dehydrogenase, almost completely abolished the effect of 18 mM ethanol on pyridoxal phosphate release and hepatic pyridoxal phosphate content of perfused organs. These results indicate that the effects of ethanol on hepatic vitamin B_6 metabolism in the rat depend upon its oxidation.

4013 BILE ACIDS. XLV. METABOLISM OF 3β,7α-DIHYDROXY-5α-CHOLESTANOIC ACID IN THE RAT WITH A BILE FISTULA. *(E.)* Mui, M. M.; Elliot, W. H. (St. Louis Univ. Sch. Med., Mo.). *Biochim. Biophys. Acta* 388(1):126-134, 1975.

The metabolism of ^{3}H-labeled 3β,7α-dihydroxy-5α-cholestanoic acid was studied in three adult male rats with cannulated bile ducts following i.p. administration of 0.34 mg of the potassium salt. Bile collected in the first 24 hr, containing 97% of the administered ^3H, was hydrolyzed and the free bile acids were separated by acetic acid partition chromatography. Of the chromatographed tritium, 58% was associated with allochenodeoxycholic acid and 14% with its 3β-isomer; only 5% of the ^{3}H was found in alcocholic acid and 1% with its 3β-isomer. Less than 5% of the tritium appeared in fractions associated with the substrate or more polar unidentified materials. Thus, this dihydroxy-5α-cholestanoic acid is metabolized in the rat primarily to dihydroxy-5α-cholanic acids, comparable to the metabolism of 3α,7α-dihydroxy-5β-cholestanoic acid in man.

4014 IMMUNOLOGICAL PROPERTIES OF TWO FETUS-SPECIFIC GLOBULINS OF RAT IN EXPERIMENTALLY INDUCED HEPATIC LESIONS. *(E.)* Watanabe, A.; TAketa, K.; Kosaka, K. (Okayama Univ. Med. Sch., Japan). *Acta Med. Okayama* 28(5):321-322, 1974.

Two fetus-specific globulins have been detected in fetal rat serum by using specific antiserum prepared from immunized rabbits. The two globulins, α_1- and α_2-fetoglobulin, migrated on polyacylamide gel discs at rates relative to bromophenol blue of 0.58 and 0.55, resp. α_2-Fetoglobulin was PAS-positive. Both α_1- and α_2-fetoglobulins were detected in amniotic fluid and serum from cadmium chloride-treated rats by immunoelectrophoresis and double-diffusion analysis. Both globulins were detected in serum from adult rats bearing dimethylaminoazobenzene-induced hepatoma. α_2-Fetoglobulin was found in serum from CCl_4, thioacetamide and cadmium chloride-treated adult rats as well as partially hepatectomized animals and rats bearing ascites hepatoma. These results suggest that α_2-fetoglobulin is a sensitive index of tissue injury.

4015 STUDIES ON LIVER REGENERATION. DEOXYRIBONUCLEIC ACID SYNTHESIS IN THE ISOLATED PERFUSED LIVER AFTER IN VITRO AND IN VIVO PARTIAL HEPATECTOMY. *(E.)* Grandjean, C.; Garcia, H. (Univ. Nebraska Med. Cent., Omaha). *Lab. Invest.* 32(4): 552-554, 1975.

DNA synthesis was studied in isolated perfused male Wistar rat livers following *in vitro* partial hepatectomy. Perfused livers were allowed to incorporate ^{3}H-thymidine for 2 hr beginning 30 min after excision of the median lobe. Specific activity of DNA from perfused regenerating livers was increased 2.2-fold compared to nonregenerating perfused livers. The rate of increase in specific activity was similar to that for *in vivo* partially hepatectomized regenerating organs (2.6-fold). These results suggest that similar regulatory mechanisms for liver regeneration

are operative both *in vitro* and *in vivo* and indicate that the liver *per se* may be the source and target of any initiating factor(s) responsible for the phenomenon.

4016 BIOSYNTHESIS OF BLOOD SERUM PROTEINS AND TRANSCRIPTION OF NUCLEAR RNA WITH REGENERATION OF RAT LIVER. *(Ukr.)* Polischuk, A. S.; Korotkoruchko, V. P.; Danko, I. M. (A. V. Palladin Inst. Biochem., Kiev, USSR). *Ukr. Biokhim. Zh.* 46(3):324-327, 1974.

4017 THE EFFECT OF THYROXINE ON ENZYME ACTIVITY IN MITOCHONDRIA OF REGENERATING RAT LIVER. *(Rus.)* Kaliman, P. A. (Kharkov Med. Inst., USSR). *Ukr. Biokhim. Zh.* 46(1):46-50, 1974.

4018 TIME CORRELATION BETWEEN DNA AND HISTONE SYNTHESIS IN REGENERATING RAT LIVER. *(Rus.)* Smirnova, V. N.; Rodinov, V. M. (Inst. Biol. Med. Chem., Moscow, USSR). *Biokhimiia* 39(6):1264-1269, 1974.

4019 EARLY AND LATE CHANGES IN NONHISTONE CHROMATIN PROTEINS ACCOMPANYING RAT LIVER REGENERATION. *(E.)* Yeoman, L. C.; Taylor, C. W.; Jordan, J. J.; Busch, H. (Baylor Coll. Med., Houston, Tex.). *Cancer Res.* 35(5):1249-1255, 1975.

4020 INDUCED CELL-CYCLE SYNCHRONY OF HEPATOCYTES AFTER PARTIAL HEPATECTOMY. *(E.)* Rabes, H. M.; Iseler, G.; Tuczek, H. V. (Inst. Pathol., Univ. Munich, Germany). *Naturwissenschaften* 62(3):142, 1975.

4021 ORNITHINE DECARBOXYLASE ACTIVITY IN REGENERATING LIVER FROM RATS ADAPTED TO CONTROLLED FEEDING SCHEDULES. *(E.)* Barbiroli, B.; Moruzzi, M. S.; Tadolini, B.; Monti, M. G. (Inst. Biol. Chem., Univ. Bologna, Italy). *J. Nutr.* 105(4):408-412, 1975.

4022 RATE OF RNA TURNOVER IN THE LIVER DURING HYPERCAPNIA IN ANIMALS. *(Ukr.)* Mykhailovsky, V. O.; Guly, M. F.; Sylonova, N. V.; Melnychuk, D. O. (O. V. Palladin Inst. Biochem., Acad. Sci. Ukr. SSR, Kiev, USSR). *Ukr. Biokhim. Zh.* 46(1):84-87, 1974.

4023 NUCLEIC ACIDS METABOLISM IN HEPATOMA 46 AND NORMAL LIVER. *(Rus.)* Bogdanov, G. N.; Shmonina, V. M. (Inst. Chem. Phys., Acad. Sci. USSR., Chernogolovka). *Ukr. Biokhim. Zh.* 46(2):181-184, 1974.

4024 HETEROTOPIC RIGHT TRANSPLANTATION OF THE LIVER WITH TOTAL PORTALIZATION IN SWINE: 124 EXPERIMENTS. *(Fr.)* Pouyet, M.; Berard, P.; Baulieux, J.; Berard, J.; Cret, R.; Magnin, G. (Claude Bernard Clin., Oullins, France). *Lyon Chir.* 70(6):383-387, 1974.

4025 STUDIES ON ORTHOTOPIC LIVER HOMOTRANSPLANTATION: THE DEVELOPMENT OF LIVER PRESERVATION APPARATUS AND LONG-TERM STORAGE OF LIVER FOR TRANSPLANTATION. *(Jpn.)* Nakashima, H. (Nara Med. Univ., Japan). *J. Nara Med. Assoc.* 25(1/2):55-71, 1974.

4026 STUDIES ON ORTHOTOPIC CANINE LIVER HOMOTRANSPLANTATION: ORTHOTOPIC HOMOTRANSTPLANTATION OF CADAVER LIVER AND TEST FOR LIVER VIABILITY PRIOR TO TRANSPLANTATION. *(Jpn.)* Oshita, Y. (Nara Med. Univ., Japan). *J. Nara Med. Assoc.* 24(6):609-620, 1974.

4027 ACTIVE ENHANCEMENT OF CANINE LIVER ALLOGRAFTS - SIMULTANEOUS PRETREATMENT WITH DONOR-SPECIFIC SEMISOLUBLE ANTIGEN AND PRENISOLONE. *(Ger.)* Lie, T. S.; Nakano, H.; Ebata, H.; Moritz, B. (Surg. Clin., Univ. Bonn., Germany). *Z. Immunitaetsforsch.* 148(1):62-72, 1974.

4028 EXTRACORPOREAL PERFUSION OF HEPATIC ALLOGRAFT AND XENOGRAFT IN ACUTE LIVER FAILURE. *(Georg.)* Bokeriia, R. I.; Dumbadze, G. G.; Dugladze, D. I.; Gaugua, A. M.; Kerdzaia, A. O.; Dzhorbenadze, T. A.; Bagrationi, E. L.; Tevdoradze, L. Sh.; Tsitsishvili, Ts. D.; Machavariani, M. I. *Soobshch. Acad. Nauk Gruzinsk. SSR* 75(2):485-488, 1974.

4029 MICROSURGICAL TECHNIQUE OF ISOLATION AND INCLUSION OF A RAT LIVER TRANSPLANT FOR HOMOGENEOUS BLOOD PERFUSION. *(Rus.)* Toguzov, R. T.; Lopukhin, Y. M.; Kukushkin, G. S. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Eksp. Khir. Anesteziol.* (4):21-23, 1974.

4030 FUNCTIONAL AND MORPHOLOGICAL CHANGES IN DONOR LIVERS AFTER VARIOUS DEGREES OF COOLING. *(Pol.)* Rozental, R. L.; Kriwulis, D. B.; Markow, J. G. (Inst. Med., Riga, USSR). *Pol. Przegl. Chir.* 46(11):1525-1530, 1974.

4031 THE USE OF AN ISOLATED LIVER TRANSPLANT IN TREATMENT OF EXPERIMENTAL HEPATIC COMA. *(Rus.)* Patsiora, M. D.; Ctetsyuk, A. G.; Farberova, I. Z.; Sholts, N. F.; Shortsinger, A. G. (Inst. Exp. Clin. Surg., Moscow, USSR). *Eksp. Khir. Anesteziol.* (4):24-28, 1974.

4032 STUDIES IN ARTERIAL CIRCULATION IN RAT LIVER. *(Ger.)* Haller, C. V. (Maingau Hosp., Frankfurt/Main, Germany). *Res. Exp. Med. (Berl).* 164(1):83-88, 1974.

4033 INTRAHEPATIC ARTERIAL MICROCIRCULATION. CLINICAL AND PATHOLOGIC FINDINGS, PHYSIOPATHOLOGY, AND TREATMENT. *(Fr.)* Isman, H. (St. Roch Hosp., Nice, France). *J. Chir. (Paris)* 107(2):105-116, 1974.

4034 SOME ASPECTS OF THE CONTROL OF HOMEOSTASIS IN ADULT LIVER. *(Fr.)* Frayssinet, C.; Moule, Y. (Inst. Sci. Res. Cancer, Villejuif, France). *J. Physiol. (Paris)* 69(Suppl.):77A-132A, 1974.

4035 THE SEQUELAE OF TEMPORARY EXPERIMENTAL OBSTRUCTION OF HEPATIC CIRCULATION. *(Rus.)* Andreiman, L. A.; Markov, J. G.; Sondor, A. A.; Kvitsinskaia, E. A. (Riga Med. Inst., USSR). *Vestn. Khir.* 113(10):54-58, 1974.

4036 DETERMINATION OF THE ORNITHINE CARBAMOYLTRANSFERASE ACTIVITY UNDER TEMPORARY OCCLUSION OF HEPATIC PORTAL VESSELS. *(Rus.)* Pogorelov, I. F. (Odessa Med. Inst., USSR). *Vopr. Med. Khir.* 20(5):523-525, 1974.

4037 HEMODYNAMICS OF EXPERIMENTAL PORTAL VENOUS OCCLUSION IN DOGS. *(E.)* Kerr, J. C.; Reynolds, D. G.; Swan, K. G. (Walter Reed Army Med. Cent., Washington, D. C.). *Am. Surg.* 41(4):198-202, 1975.

4038 PORTACAVAL ANASTOMOSIS IN THE RAT. V. HISTOLOGICAL CHANGES. *(It.)* Cherubini, M.; Silvestri, F.; Milone, E.; Pettirossi, O.; Di Pietrantonio, M. (Inst. Surg. Clin., Univ. Trieste, Italy). *Policlinco [Chir.]* 81(3):144-156, 1974.

4039 EXPERIMENTAL STUDY OF POSTHEPATIC LYMPHODYNAMICS DURING PORTAL HYPERTENSION. *(It.)* Anfossi, A.; Cavaliere, P.; Adami, G. F.; Tallero, G.; Gianetta, E. (Inst. Gen. Clin. Surg., Surg, Ther., Univ. Genoa, Italy). *Chir. Patol. Sper.* 21(4):274-284, 1973.

4040 ARTERIALIZATION OF THE LIVER IN COMBINATION WITH A PORTACAVAL SHUNT IN THE DOG. *(E.)* Adamsons, R. J.; Arif, S.; Babich, A.; Butt, K.; Lam, A.; Minkowitz, S. (Downstate Med. Cent., State Univ. New York, Brooklyn). *Surg. Gynecol. Obstet.* 140(4):594-600, 1975.

4041 ELECTRON MICROSCOPE EXAMINATION OF THE SITE OF ACID PHOSPHATASE INSIDE THE LYSOSOMES. *(Rus.)* Pokrovskii, A. A.; Krystev, L. P.; Tutelian, V. A.; Boiadzhieva, Zh. K.; Kravchenio, L. V. (Inst. Nutr., Moscow, USSR). *Dokl. Acad. Nauk SSSR* 219(5):1235-1237, 1974.

4042 ELECTROPHORETIC ANALYSIS OF HISTONES IN THE HEPATIC CELL NUCLEI IN CALORIC-DEFICIENT ALBINO RATS. *(Rus.)* Klimenko, A. I.; Khints, R. (Inst. Biol., A.M. Gorky State Univ., Kharkov, USSR). *Vopr. Pitan.* (6):16-19, 1974.

4043 STEROL CARRIER PROTEIN HYPOTHESIS: REQUIREMENT FOR THREE SUBSTRATE-SPECIFIC SOLUBLE PROTEINS IN LIVER CHOLESTEROL BIOSYNTHESIS. *(E.)* Scallen, T. J.; Seetharam, B.; Srikantaiah, M. V.; Hansbury, E.; Lewis, M. K. (Dep. Biochem., Univ. New Mexico, Albuquerque). *Life Sci.* 16(6):853-874, 1975.

4044 REGULATION OF LIPOGENESIS IN ANIMALS. *(Ger.)* Numa, S. (Fac. Med., Kyoto Univ., Japan). *Naturwissenschaften* 62(2):80-86, 1975.

4045 DISTRIBUTION AND TRANSPORT OF [^{14}C] CHOLESTEROL BETWEEN ORGANELLAS OF CELLS IN RAT LIVER. *(Rus.)* Kolomiytseva, I. K.; Kaznacheev, Y. S. (Inst. Biol. Phys., Acad. Sci. USSR, Pushchino). *Ukr. Biokhim. Zh.* 46(6):777-781, 1974.

4046 EFFECT OF FASTING ON THE FREE FATTY ACID CONTENT OF THE LIVER AND KIDNEY IN THE RAT. *(Cz.)* Chorvathova, V; Ondreicka, R.; Ovecka, M. (Res. Inst. Hum. Nutr. Bratislava, Czechoslovakia). *Cz. Gastroent. Vyz.* 28(7):461-466, 1974.

4047 THE INFLUENCE OF A HIGH LEVEL OF DIETARY CORN OIL ON RAT SERUM AND LIVER LIPIDS. *(E.)* Narayan, K. A.; McMullen, J. J.; Butler, D. P.; Wakefield, T.; Calhoun, W. K. (Nutr. Div., US Army Natick Lab., Mass.). *Nutr. Reports Int.* 10(1):25-34, 1974.

4048 SIMPLIFIED METHODS FOR THE EXAMINATION OF LIVER LIPIDS. I. DETERMINATION OF VITAMIN A IN LIVER USING A DIRECT SOLVENT EXTRACTION TECHNIQUE. *(E.)* Bayfield, R. F. (Vet. Res. Stn., Dep. Agric., Glenfield, Australia). *Anal. Biochem.* 64(2):403-413, 1975.

4049 METABOLISM OF ETHANOL. I. HEPATIC OXIDATION OF ETHANOL AND ITS BIOCHEMICAL EFFECT ON THE LIVER. *(Fr.)* Gajdos, A. (Lab. Biochem. Res., Rene Descartes Univ., Paris, France). *Ann. Biol. Clin.* 32(5):405-411, 1974.

4050 EFFECT OF ETHANOL ON UREA SYNTHESIS FROM AMMONIA IN ISOLATED LIVER CELLS FROM FED AND STARVED RATS. *(E.)* Williamson, J. R.; DeLeeuw, G.; Meijer, A. J. (Johnson Res. Found., Univ. Pennsylvania, Philadelphia). *Fed. Proc.* 34(3):573, 1975.

4051 DRUG METABOLISM STUDIES IN ISOLATED RAT HEPATOCYTES. *(E.)* Billings, R. E.; Ashmore, J.; Wagle, S. R.; McMahon, R. E. (Indiana Univ. Sch. Med., Indianapolis). *Fed. Proc.* 34(3):727, 1975.

4052 EVIDENCE LINKING SINGLET OXYGEN FORMATION WITH DRUG METABOLISM AND LIPID PEROXIDATION IN RAT LIVER MICROSOMES. *(E.)* King, M.; Lai, E.; McCay, P. B. (Oklahoma Med. Res. Found., Oklahoma City). *Fed. Proc.* 34(3):622, 1975.

4053 INFLUENCE OF PARTIAL DEHYRATION ON THE QUANTITATIVE CYTOARCHITECTURE OF RAT HEPATOCYTES (A CYTOPHOTOMETRIC AND MORPHOMETRIC STUDY). *(Ger.)* Riede, U. N.; Kreutzer, W.; Robausch, T.; Kiefer, G.; Sandritter, W. (Pathol. Inst., Univ. Freiburg; Germany). *Beitr. Pathol.* 153(4):379-394, 1974.

4054 ARE ATYPICAL LIVER MITOCHONDRIA SPECIAL ADAPTIVE FORMS? III. PARACRYSTALLINE INCLUSIONS IN THE INTRACRISTAL SPACE. *(Ger.)* Riede, U. N.; Nobmann, E. (Pathol. Inst., Univ. Freiburg, Germany). *Beitr. Pathol.* 153(4):319-329, 1974.

4055 EFFECT OF RENAL MICROSOMES AND RENAL LYSOSOMES ON IN VITRO HEPATIC FATTY ACID SYNTHESIS. *(E.)* Haven, G. T.; Nguyen, T. T.; Krzemien, J. R.; Thuy, L. P. (Coll. Med., Univ. Nebraska, Omaha). *Res. Commun. Chem Pathol. Pharmacol.* 10(3): 473-490, 1975.

4056 THE DEVELOPMENT OF KINETIC PARAMETERS OF HEPATIC DRUG-METABOLIZING ENZYMES IN PERINATAL RATS. *(E.)* Bell, J. U.; Ecobichon, D. J. (Dep. Pharmacol., Dalhousie Univ., Halifax, Canada). *Can. J. Biochem.* 53(4):433-437, 1975.

4057 INHIBITION OF NADPH OXIDATION AND OXIDATIVE METABOLISM OF DRUGS IN LIVER MICROSOMES BY ZINC. *(E.)* Chvapil, M; Sipes, I. G.; Ludwig, J. C.; Halladay, S. C. (Univ. Arizona Med. Sch., Tucson). *Biochem. Pharmacol.* 24(8):917-919, 1975.

4058 CADMIUM POTENTIATION OF DRUG RESPONSE - ROLE OF THE LIVER. *(E.)* Johnston, R. E.; Miya, T. S.; Schnell, R. C. (Sch. Pharm. Pharm. Sci., Purdue Univ., West Lafayette, Ind.). *Biochem. Pharmacol.* 24(8):877-881, 1975.

4059 EFFECT OF SILYBIN ON THE HEPATIC MICROSOMAL DRUG-METABOLISING ENZYME SYSTEM IN THE RAT. *(E.)* Down, W. H.; Chasseaud, L. F.; Grundy, R. K. (Huntingdon Res. Cent., England). *Arzneim. Forsch.* 24(12):1986-1988, 1974.

4060 PROLIFERATION KINETICS OF BILE DUCT EPITHELIA IN THE REGENERATING MOUSE LIVER AFTER CCl_4 POISONING. *(Ger.)* Gerhard, H.; Schultze, B.; Maurer, W. (Inst. Med. Radiol., Univ. Wurzburg, Germany). *Virchows Arch.* [*Zellpathol.*] 17(3):213-227, 1975.

4061 HEXOKINASE ISOZYME PATTERN IN CCl_4-INJURED RAT LIVER. *(E.)* Ueda, M.; Taketa, K.; Kosaka, K. (Okayama Univ. Med. Sch., Japan). *Clin. Chim. Acta.* 60(1):77-84, 1975.

4062 DETECTION OF EARLY METABOLITES IN RAT LIVER AFTER ADMINISTRATION OF CCl_4 AND $CBrCl_3$. *(E.)* Bini, A.; Vecchi, G.; Vivoli, G.; Vannini, V.; Cessi, C. (Inst. Gen. Pathol., Univ. Modena, Italy). *Pharmacol. Res. Commun.* 7(2):143-149, 1975.

4063 SYNTHESIS OF RAT LIVER RNA UNDER CONDITIONS OF PROTEIN SYNTHESIS INHIBITION BY CYCLOHEXIMIDE. *(Ukr.)* Todorov, I. N.; Halkin, A. P.; Kornyushyna, T. V.; Yuhai, N. V.; Hovze, Y. H. (Inst. Mol. Biol. Genet., Acad. Sci. Ukr. SSR, Kiev, USSR). *Ukr. Biokhim. Zh.* 46(4):431-437, 1974.

4064 INFLUENCE OF LEAD-OVERLOAD ON THE QUANTITATIVE CYTOARCHITECTURE OF JUVENILE RAT HEPATOCYTES. *(Gen.)* Riede, U. N.; Rasser, Y. M.; Rohr, H. P. (Inst. Pathol., Univ. Basel, Switzerland). *Beitr. Pathol.* 152(4):383-394, 1974.

4065 GLUCOCORTICOID INDUCED LESIONS IN RAT LIVERS. *(E.)* Kim, Y. S.; Choi, B. H.; Kim, Y. (St. Louis Univ. Sch. Med., Mo.). *Exp. Mol. Pathol.* 22(2):260-270, 1975.

4066 IN VIVO DETOXIFICATION OF ENDOTOXIN BY MOUSE LIVER. *(E.)* Rippe, D. F.; Soltas, J. G.; Berry, L. J. (Dep. Microbiol., Univ. Texas, Austin). *J. Reticuloendothel. Soc.* 16(3):175-183, 1974.

4067 LIVER LEVELS OF α-GLYCEROPHOSPHATE, GLYCEROPHOSPHATE DEHYDROGENASE AND ACETYL-CoA CARBOXYLASE DURING PHENOBARBITAL INDUCTION. *(E.)* Depierre, J. W.; Ernster, L. (Dep. Biochem., Arrhenius Lab., Univ. Stockholm, Sweden). *FEBS Lett.* 52(2)L 240-241, 1975.

4068 FACTORS INVOLVED IN INCREASED PROTEIN SYNTHESIS IN LIVER MICROSOMES AFTER ADMINISTRATION OF DDT. *(E.)* Cappon, I. D.; Nicholls, D. M. (Dep. Biol., York Univ., Toronto, Canada). *Pestic. Biochem. Physiol.* 5(2):109-118, 1975.

4069 INFLUENCE OF PARENTERAL ZINC AND ACTINOMYCIN D ON TISSUE ZINC UPTAKE AND THE SYNTHESIS OF A ZINC-BINDING PROTEIN. *(E.)* Richards, M. P.; Cousins, R. J. (Dep. Nutr., Cook Coll., Rutgers Univ., New Brunswick, N.J.). *Bioinorg. Chem.* 4(3):215-224, 1975.

4070 2-AMINOACETAMIDO-N-ARYLBENZAMIDES AS POTENTIAL CHOLERETIC AND ANTIFIBRILLATORY AGENTS. *(E.)* Bonola, G.; Magistretti, M. J.; Setnikar, I.; Massarani, E. (Res. Div., Recordati S.p. A., Milan, Italy). *Eur. J. Med Chem.* 7121(9):639-640, 1974.

4071 BIOLOGICAL POTENCY OF ORGANIC SELENIUM COMPOUNDS: VI. ALIPHATIC SELENINIC ACIDS AND CARBOXYSELENINIC ACIDS. *(E.)* Schwarz, K.; Fredga, A. (VA Hosp., Long Beach, Calif.). *Bioinorg. Chem.* 4(3):235-243, 1975.

4072 CHANGES IN METABOLISM OF ETHANOLAMINE AND ITS DERIVATIVES IN LIVER DURING FASTING. *(E.)* Haines, D. S. M.; Derksen, D. H. (Victoria Hosp., London, Canada). *Can. J. Biochem.* 53:51-56, 1975.

4073 CELLULAR REACTION OF HEPATIC STROMA IN VARIOUS REGIMENS OF PATHOGENIC ACTION. *(Rus.)* Popova, I. V. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 78(12):102-105, 1974.

4074 THE EFFECT OF Mg^{++} IONS ON LIVER PRESERVATION. *(Ger.)* Lie, T. S.; Nakano, H.; Haupt, H.; Ebata, H.; Rommelsheim, K. (Surg. Clin., Univ. Bonn, Germany). *Bruns Beitr. Klin. Chir.* 221(8):643-646, 1974.

4075 THE ALTERATIONS IN CoA CONTENT IN ORGANS OF YOUNG RATS AFTER ADMINISTRATION OF PANTOTHENATE DERIVATIVES. *(Rus.)* Rosanov, A. Y.; Agafonova, N. Y.; Yakovlev, V. A.; Kopelevich, V. M.; Yevdokimova, G. S. (I. I. Mechnikov State Univ., Odessa, USSR). *Vopr. Med. Khim.* 20(4):363-367, 1974.

4076 ACCUMULATION OF MALONYLDIALDEHYDE IN ISOLATED RAT LIVER. *(Rus.)* Panchenko, L. F.; Gerasimov, A. M.; Nozdrachova, L. I.; Kariakina, G. A. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Vopr. Med. Khim.* 20(3):321-324, 1974.

4077 THE EFFECT OF HYDROCORTISONE AND ACUTE HYPOXIA ON GLUCONEOGENESIS OF RAT LIVER. *(Rus.)* Postupaev, V. V.; Litonyan, Z. M. (Khabarovsk Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 78 (12):19-24, 1974.

4078 EFFECT OF VITAMIN E ON INTENSITY OF METHIONINE TRANSFORMATION IN RAT LIVER. *(Ukr.)* Shtutman, T. M.; Biber, H. O. (A. V. Palladin Inst. Biochem., Kiev, USSR). *Ukr. Biokhim. Zh.* 46(2): 192-196, 1974.

4079 CYTOFLUOROMETRIC STUDY OF THE TWO GLYCOGEN FRACTIONS IN THE RAT LIVER CELLS. *(Rus.)* Kudryavtseva, M. V.; Kudryavtsev, B. N.; Rosanov, Y. M. (Inst. Cytol., Acad. Sci. USSR, Leningrad). *Cytologiia* 16(7):851-858, 1974.

4080 CYCLIC NUCLEOTIDES AND GLUCONEOGENESIS BY RAT LIVER CELLS. *(E.)* Fain, J. N.; Tolbert, M. E. M.; Pointer, R. H.; Butcher, F. R.; Arnold, A. (Div. Biol. Med. Sci., Brown Univ., Providence, R.I.). *Metabolism* 24(3):395-408, 1975.

4081 SEX DIFFERENCES IN THE EFFECTS OF ETHIONINE ON RNA AND PROTEIN SYNTHESIS IN SWISS MICE. *(E.)* Berry, D. E.; Friedman, M. A. (Med. Col. Virginia, Richmond). *Fed. Proc.* 34(3):872, 1975.

4082 INFLUENCE OF DL-ETHIONINE ON THE ACCUMULATION AND SUBCELLULAR DISTRIBUTION OF COPPER IN THE LIVER OF RATS INJECTED WITH COPPER. *(E.)* Balevska, P. S. (Inst. Physiol., Bulgarian Acad. Sci., Sofia). *C. R. Acad. Sci. (Bulg.)* 28(1): 125-127, 1975.

4083 NEONATAL IMPRINTING AND THE TURNOVER OF MICROSOMAL CYTOCHROME P-450 IN RAT LIVER. *(E.)* Levin, W.; Ryan, D.; Kuntzman, R.; Conney, A. H. (Dep. Biochem. Drug Metab., Hoffmann-La Roche, Inc., Nutley, N.J.). *Mol. Pharmacol.* 11(2):190-200, 1975.

4084 NOVEL LIVER MICROSOMAL ENZYME INHIBITORS. *(E.)* Israili, Z. H.; Black, N. B.; Dayton, P. G. (Dep. Med., Emory Univ., Atlanta, Ga.). *Fed. Proc.* 34(3):784, 1975.

4085 STUDIES ON ACUTE PHASE PROTEINS OF RAT SERUM. V. EFFECT OF INDUCED INFLAMMATION ON THE SYNTHESIS OF ALBUMIN AND α_1-ACID GLYCOPROTEIN BY LIVER SLICES. *(E.)* Jamieson, J. C.; Morrison, K. E.; Molasky, D.; Turchen, B. (Dep. Chem., Univ. Manitoba, Winnipeg, Canada). *Can. J. Biochem.* 53(4): 401-414, 1975.

4086 THIAMINE INCREASES THE SPECIFIC ACTIVITY OF HUMAN LIVER BRANCHED CHAIN α-KETOACID DEHYDROGENASE. *(E.)* Danner, D. J.; Davidson, E. D.; Elsas II, L. J. (Emory Univ. Sch. Med., Atlanta, Ga.). *Nature* 254(5500):529-530, 1975.

4087 INCORPORATION OF AMINO ACIDS INTO PROTEINS OF RAT LIVER SUBCELLULAR FRACTIONS UNDER THE EFFECT OF MULTIPLE INJECTIONS OF ACTH AND ACTH COMBINATION WITH RNA. *(Ukr.)* Hermaniuk, Ia. L.; Sydorenko, D. S. (Kiev Sci. Res. Inst. Endocrin. Metabol., Min. Public Health Ukr. SSR, Kiev, USSR). *Ukr. Biokhim. Zh.* 46(4):438-440, 1974.

4088 EFFECTS OF FASTING ON THE ACTIVITY OF CYTOSOL FACTORS ENGAGED IN PROTEIN BIOSYNTHESIS IN MOUSE HEPATOCYTES. *(E.)* Gamulin, S.; Norman, M. R. (King's Coll. Hosp. Med. Sch., London, England). *Nutr. Metabol.* 17(2):121-126, 1974.

4089 EFFECT OF MULTIPLE INJECTIONS OF ACTH, ALONE AND IN COMBINATION WITH RNA, ON PROTEIN METABOLISM IN THE LIVER, SPLEEN, AND MUSCLES OF ALBINO RATS. *(Ukr.)* Hermaniuk, Ia. L.; Sydorenko, D. S.; Bohdan, I. P. (Kiev. Res. Inst. Endocrinol. Metab., USSR). *Dopovidi Akad. Nauk Ukr. RSR [B]* (11):1036-1039, 1974.

4090 INFLUENCE EXERTED BY VITAMIN A ON THE ACTIVITY OF PHENYLALANINE-4-HYDROXYLASE AND TYROSINE-AMINOTRANSFERASE IN THE RAT LIVER. *(Rus.)* Fedurov, V. V.; Terentieva, V. G. (Kiev Med. Inst., USSR). *Vopr. Pitan.* (6):12-16, 1974.

4091 INDUCTION AND INHIBITION OF TYROSINE AMINOTRANSFERASE IN MOUSE AND RAT LIVER BY VITAMIN B_6 ANTAGONISTS AND BY INHIBITORS OF PYRIDOXAL-5'-PHOSPHATE ENZYMES. *(Rus.)* Bukin, Iu. V.; Matveeval, A. A. (Inst. Exp. Clin. Oncol., Moscow, USSR). *Biokhimiia* 39(6):1146-1156, 1974.

4092 BREAKDOWN OF ALBUMIN IN THE SUBCELLULAR COMPARTMENTS OF THE LIVER. *(Ger.)* Massarrat, S.; Siai, D. J. (Med. Polyclin., Phillips Univ., Marburg/Lahn, Germany). *Res. Exp. Med. (Berl.)* 164(1):49-58, 1974.

4093 EVIDENCE FOR A UDP-GLUCOSE RECEPTOR IN THE MICROSOMAL MEMBRANES OF RAT HEPATOCYTES. *(Fr.)* Frot-Coutaz, J.; Berthillier, G.; Got, R. (Lab. Biochem., Univ. Claude Bernard, Lyon, France). *Febs Lett.* 52(1):81-84, 1975.

4094 EVOLUTION AND CONTROL OF CARBAMYL PHOSPHATE SYNTHETASE AND ORNITHINE TRANSCARBAMYLASE IN DEVELOPING FETAL RAT LIVER. *(Fr.)* Gautier, C.; Husson, A.; Vaillant, R. (Lab. Endocrinol., Univ. Rouen, France). *Experientia* 31(1):125-126, 1975.

4095 REGULATION OF PYRUVATE DEHYDROGENASE INTERCONVERSION IN ISOLATED HEPATOCYTES BY THE MITOCHONDRIAL ATP/ADP RATIO. *(E.)* Siess, E. A.; Wieland, O. H. (Schwabing Hosp., Munich, Germany). *Febs Lett.* 52(2):226-230, 1975.

4096 CORTICOSTERONE CONTENT IN NUCLEI OF THE HEART, BRAIN, AND LIVER OF ALBINO RATS AT DIFFERENT AGES. *(Ukr.)* Nesterenko, H. A. (Inst. Biol., Kharkov State Univ., USSR). *Dopovidi Akad. Nauk Ukr. RSR* [B] (11):1042-1044, 1974.

4097 METABOLISM OF GLUCOCORTICOIDS AND THEIR TRANSPORT-RESERVE FORMS IN ISOLATED PERFUSED LIVER. *(Ukr.)* Treshchynskyi, A. I.; Mozhaev, H. A.; Shlapak, I. P. (Inst. Postgrad. Training Physicians, Kiev, USSR). *Ukr. Biokhim. Zh.* 46(2): 188-191, 1974.

4098 INFLUENCE OF THE LIVER ON THE PRE- AND POSTHEPATIC CONCENTRATIONS OF PLASMA HISTAMINE IN HUMAN SUBJECTS. *(Ger.)* Stopik, D.; Berger, H. G.; Bittner, R.; Brauer, D.; Fiedler, A. (Westend. Clin., Free Univ. Berlin, Germany). *Klin. Wochenschr.* 52(14):696-698, 1974.

4099 PROLACTIN RECEPTORS IN RAT LIVER: POSSIBLE INDUCTION BY PROLACTIN. *(E.)* Posner, B. I.; Kelly, P. A.; Friesen, H. G. (McGill Univ. Clin., Montreal, Canada). *Science* 188(4183): 57-59, 1975.

4100 A METHOD FOR PERFUSION OF THE CANINE LIVER IN VIVO VIA THE HEPATIC ARTERY WITH BLOOD OF A CONTROLLED GAS CONTENT. *(E.)* Andrews, W. H. H.; Bradley, D. J. (R. Free Hosp. Sch. Med., London, England). *J. Physiol. (Lond.)* 246(2):5P-6P, 1975.

4101 EFFECT OF THE AMMONIA TOLERANCE TEST ON ISOLATED PERFUSED SWINE LIVER. *(Ger.)* Hirner, A.; Haring, R.; Stallkamp, B; Tung, L.C.; Waldschmidt, J. (Steglitz Clin., Free Univ. Berlin, Germany). *Z. Exp. Chir.* 7(3):179-187, 1974.

4102 SELECTIVE IN SITU PERFUSION OF THE CANINE LIVER. *(Ger.)* Duca, S. (Surg. Clin. III., Cluj, Rumania). *Bruns Beitr. Klin. Chir.* 221(8):647-654, 1974.

4103 THE PATHOPHYSIOLOGY OF ACUTE RENAL INSUFFICIENCY ASSOCIATED WITH HEPATOBILIARY DISEASE. *(Ger.)* Peschl, L. (Kaiserin Elisabeth Hosp., Vienna, Austria). *Acta Med. Aust.* 1(5): 144-150, 1974.

4104 EXPERIMENTAL LYMPHOSTASIS OF THE LIVER. *(Ger.)* Lie, T. S.; Seifert, G.; Fasske, E.; Nakano, H.; Ebata, H.; Bartsch, G. (Surg. Clin. Polyclin., Univ. Bonn, Germany). *Leber Magen Darm* 4(6):313-316, 1974.

4105 THE INHIBITION OF THE UPTAKE OF ^{3}H-THYMIDINE INTO LIVER DNA BY THE INTRAPORTAL INFUSION OF FRESH SERUM AFTER PARTIAL HEPATECTOMY IN THE RAT. *(E.)* Bradbrook, R. A.; Newcombe, R. G.; Thatcher, J.; Blumgart, L. H. (Welsh Natl. Sch. Med., Cardiff). *Eur. Surg. Res.* 6(6):364-374, 1974.

4106 EXPERIMENTAL HEPATOGRAPHY WITH IOPHENDYLATE. *(Rus.)* Sviridov, N. K. (Moscow Res. Inst. Roentgenol. Radiol., USSR). *Eksp. Khir. Anesteziol.* (5):53-55, 1974.

4107 HEPATOPROTECTIVE ACTION OF DICARBOXYLIC AMINO ACIDS AND AMIDES. *(Rus.)* Maliugin, E. F.; Khokhlov, A. P.; Terentieva, V. B.; Gogiberidze, G. V. (N.I. Pirogov 2nd Moscow Med. Inst., USSR). *Farmakol. Toksikol.* 37(6):703-704, 1974.

4108 THERAPEUTIC ATTEMPT IN EXPERIMENTAL HEPATITIS. *(Ser.)* Stern, P.; Gmaz-Nikulin, E. (Inst. Pharmacol., Fac. Med., Sarajevo, Yugoslavia). *Acta Med. Iugosl.* 28(5):447-451, 1974.

4109 CHANGE IN FUNCTION OF OVARIES AND ADRENAL GLANDS IN RATS WITH EXPERIMENTAL TOXIC HEPATITIS. *(Rus.)* Khominskaia, Z. B. (Inst. Pediatr., Gynecol., Obstet., Kiev, USSR). *Fiziol. Zh.* 20(6):747-751, 1974.

4110 DISRUPTION OF THE HETEROPHAGE FUNCTION OF LIVER LYSOSOMES IN RATS SUFFERING FROM ACUTE AND CHRONIC TOXIC HEPATITIS. *(Rus.)* Korolenko, T. A.; Titova, V. G.; Kondakova, A. E. (Novasibirsk Med. Inst., USSR). *Farmakol. Toksikol.* 37(6):725-727, 1974.

4111 BIOCHEMICAL AND MORPHOLOGICAL CHANGES IN THE LIVER AND BLOOD SERUM IN EXPERIMENTAL LIVER FAILURE. *(Rus.)* Rusakov, V. I.; Alimova, E. K.; Lokshin, L. S.; Pustovalova, L. M.; Dzarokhokhov, K. I. (Rostov Med. Inst., USSR). *Eksp. Khir. Anesteziol.* (6):41-44, 1974.

4112 CHANGE IN SUBMICROSCOPIC ORGANIZATION OF HEPATOCYTES, AND IN HEPATOCYTE AND CATION CONTENTS UNDER EXPERIMENTAL RACHITES IN RATS. *(Ukr.)* Mynonova, V. M.; Khomutovsky, O. A. (A. V. Palladin Inst. Biochem., Kiev, USSR). *Ukr. Biokhim. Zh.* 46(5):635-638, 1974.

4113 CONTENT AND DISTRIBUTION OF UBIQUINONE AND UBICHROMENOL IN UNSAPONIFIABLE FRACTION OF LIVER SUBCELLULAR STRUCTURES IN NORMAL RATS AND RATS WITH HYPOVITAMINOSIS A AND E. *(Ukr.)* Chernukhina, L. O.; Donchenko, G. V.; Kovalenko, V. M. (A. V. Palladin Inst. Biochem., Kiev, USSR). *Ukr. Biokhim. Zh.* 46(4):514-518, 1974.

4114 CHANGES OF SPECIFIC ACTIVIY AND SYNTHESIZING ACTIVITY OF CATALASE IN ACATALASEMIA MOUSE LIVER. *(E.)* Ogata, M.; Takagi, M.; Ogata, K.; Tomokuni, K.; Mizugaki, J.; Takahara, S. (Okayama Univ. Med. Sch., Japan). *Tohoku J. Exp. Med.* 114 (4):349-354, 1974.

4115 GALACTOSAMINE-"HEPATITIS" IN RATS WITH DIFFERENT PHAGOCYTIC ACTIVITY OF LIVER RES. *(E.)* Rasenack, U.; Grun, M.; Liehr, H. (Dep. Int. Med., Univ. Wurzburg, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):29-35, 1975.

4116 EXCRETION OF BILIRUBIN IN THE GASTROINTESTINAL TRACT AND ITS CONVERSION INTO UROBILINOGEN. THE ENTEROHEPATIC CIRCULATION AND EXCRETION OF UROBILINOGEN. DIRECT TRANSPORT BETWEEN THE BLOOD AND GASTROINTESTINAL TRACT. *(It.)* Coltorti, M.; Di Somone, A. (1st Fac. Med. Surg., Univ. Naples, Italy). *Fegato* 19(2):167-204, 1973.

4117 TRANSPORT OF BILIRUBIN FROM BLOOD PLASMA TO HEPATOCYTES. *(It.)* Frezza, M.; Tiribelli, C. (Inst. Spec. Med. Pathol., Univ. Trieste, Italy). *Fegato* 19(2):109-117, 1973.

4118 CHANGES IN BILIRUBIN IN THE HEPATOCYTE AND IN EXTRAHEPATIC SITES. *(It.)* Michelazzi, L.; Nanni, G.; Marinari, U. M.; Casu, A. (Inst. Gen. Pathol., Univ. Genoa, Italy). *Fegato* 19(2): 119-144, 1973.

4119 INTERACTIONS BETWEEN BILIRUBIN AND COMPONENTS OF THE HEPATOCYTE MEMBRANE. *(It.)* Casu, A.; Pala, V.; Marinari, U. M.; Nanni, G. (Inst. Gen. Pathol., Univ. Genoa, Italy). *Fegato* 19(2): 145-159, 1973.

4120 INTERACTIONS BETWEEN THE EXCRETION OF BENZYLPENICILLIN, BILIRUBIN AND BSP IN THE BILE: EFFECT OF SOME LIVER FRACTIONS AND THEIR CONSTITUENTS. *(It.)* Paroli, E.; Samueli, F.; Valeri, P. (Dep. Pharmacol., Univ. Rome, Italy). *Fegato* 19(2):293-300, 1973.

4121 SUBCELLULAR CARBOXYLESTERASE ACTIVITY AND THE HEPATIC TRANSPORT OF BILIRUBIN. *(It.)* Burlina, A.; Boccato, P.; Sorgato, K.; Galzigna, L. (Gen. Provinc. Hosp., Conegliano, Italy). *Fegato* 19(2):161-166, 1973.

4122 INTERACTION OF BILIRUBIN WITH HUMAN ERYTHROCYTE MEMBRANE. *(E.)* Kapoor, C. L. (Cent. Drug Res. Inst., Lucknow, India). *Curr. Sci.* 44(3):82-84, 1975.

4123 SUBSTANCES INTERFERING WITH BILIRUBIN TRANSPORT IN THE BLOOD BY COMPETING WITH ALBUMIN: SUBSTANCES INHIBITING THE UPTAKE OF BILIRUBIN BY HEPATOCYTES. *(It.)* Periti, P.; Serra, E. (Dep. Chemother., Univ. Florence, Italy). *Fegato* 19(2): 225-236, 1973.

4124 THE EFFECT OF PHENOBARBITAL ON THE SERUM BILIRUBIN LEVEL IN NORMAL AND CCl_4 LIVER-DAMAGED RATS. BIOCHEMICAL AND HISTOLOGIC STUDY. *(E.)* Tsironis, A. G.; Mykoniatis, M. G.; Papacharalampous, N. X. (Inst. Exp. Pharmacol., Athens Univ., Greece). *Acta Hepatogastroenterol. (Stuttg.)* 22(1): 25-29, 1975.

4125 BASIC BIOCHEMICAL AND PHYSIOLOGICAL ASPECTS OF THE METABOLISM OF BILE ACIDS. *(Ger.)* Wildgrube, H. J.; Leuschner, U. (Cent. Intern. Med., Johann-Wolfgang Goethe Univ., Frankfurt, Germany). *Leber Magen Darm* 4(1):5-12, 1974.

4126 ISOLATION OF LIVER CELLS AND IDENTIFICATION OF A POSSIBLE BILE DUCTULE CELL POPULATION. *(E.)* Grant, A. G. (R. Free Hosp., London, England). *J. Physiol. (Lond.)* 246(2):4P, 1975.

4127 RECEPTORS IN THE BILE DUCTS. *(Rus.)* Dekhkanov, T. D. (I. P. Pavlov Med. Inst., Samarkand, USSR). *Arkh. Anat. Gistol. Embriol.* 67(9): 110-113, 1974.

4128 HYDROXYLATION OF BILE ACID PRECURSORS BY RABBIT MICROSOMES. *(E.)* Elliott, W. H.; Ali, S. S. (St. Louis Univ. Sch. Med., Mo.). *Fed. Proc.* 34(3):661, 1975.

4129 AN EVALUATION OF SOME OF THE POTENTIAL IMMEDIATE SOURCES OF CHOLESTEROL FOR BILE ACID SYNTHESIS IN SWINE. *(E.)* Kim, D. N.; Lee, K. T.; Reiner, J. M.; Thomas, W. A. (Albany Med. Coll., N.Y.). *Exp. Mol. Pathol.* 22(2):284-293, 1975.

4130 DYNAMICS OF BILE ACID CONTENT IN RAT TISSUES UNDER EXPERIMENTAL OBSTRUCTIVE JAUNDICE. *(Ukr.)* Hanitevych, Y. V.; Karbach, Y. I.; Kovtun, T. D. (Dep. Hum. Anim. Physiol., Univ. Chernovtsy, USSR). *Ukr. Biokhim. Zh.* 46(5):658-660, 1974.

4131 THE ACTION OF HYDROCORTISONE ON THE DNA SYNTHESIS OF THE EPITHELIAL CELLS OF THE BILE DUCTS. *(Rus.)* Dyban, P. A. (Inst. Exp. Med., Leningrad, USSR). *Biull. Eksp. Biol. Med.* 78(12): 77-80, 1974.

4132 INFLUENCE EXERTED BY SODIUM THIOPENTAL ON BILE PRODUCTION IN THE LIVER. *(Rus.)* Skarlosh, V. M. (Ternopol Med. Inst., USSR). *Farmakol. Toksikol.* 37(6):701-702, 1974.

4133 THE EFFECTS OF CHOLECYSTECTOMY ON THE BILE SALT FOOL OF THE SYRIAN HAMSTER. *(E.)* Beher, W. T.; Lin, G. J.; Konde, W. N.; Stradnieks, S. (Henry Ford Hosp., Detroit, Mich.). *Fed. Proc.* 34(3):661, 1975.

4134 THE INFLUENCE OF GALASCORBIN ON THE COURSE OF EXPERIMENTAL ACUTE CHOLECYSTITIS IN DOGS. *(Rus.)* Shevchenko, V. S. (Kiev Med. Inst., USSR). *Eksp. Khir. Anesteziol.* (6):46-49, 1974.

4135 CHANGES IN VASCULAR-NERVE CELL RELATIONS IN THE WALL OF THE GALLBLADDER IN EXPERIMENTAL CHOLECYSTITIS. *(Rus.)* Melman, E. P.; Krinitski, S. S. (Ivano-Frankovsk Med. Inst., USSR). *Arkh. Anat. Gistol. Embriol.* 67(7):20-25, 1974.

4136 PORTACAVAL SHUNT IN THE RAT: CONSEQUENCES ON BILE FLOW AND COMPOSITION. *(It.)* Milone, E.; Pettirossi, O.; Cherubini, M.; Di Pietrantonio, M. (Inst. Clin. Surg., Univ. Trieste, Italy). *Acta Chir. Ital.* 30(3):325-331, 1974.

4137 BILE-FORMING FUNCTION OF THE LIVER IN STREPTOCOCCAL INFECTION. *(Rus.)* Porokhniak, L. A. (Inst. Pharmacol., Ternapol, USSR). *Vrach. Delo* 616(2):59-61, 1975.

4138 INFLUENCE OF EXTRAHEPATIC CHOLESTASIS ON METABOLISM AND BILIARY EXCRETION OF IMIPRAMINE. *(E.)* Gigon, P. L. (Inst. Med. Chem., Univ. Bern, Switzerland). *Arch. Int. Pharmacodyn. Ther.* 213(2):211-221, 1975.

4139 EXPERIMENTAL STENOSIS OF THE AMPULLA OF VATER. *(It.)* Farello, G. A.; Motton, G.; Fontanin, O.; De Togni, F.; Gandolfi, P. (Inst. Spec. Surg. Pathol., Univ. Padua, Italy). *Acta Chir. Ital.* 30(3):317-324, 1974.

4140 CHANGES IN THE ENZYMATIC ACTIVITY OF THE PERFUSATE UNDER CONDITIONS OF EXTRACORPOREAL CIRCULATION OF THE ISOLATED LIVER. *(Rus.)* Krivulis, D. B.; Kvitsinskaya, E. A. (Riga Med. Inst., USSR). *Patol. Fiziol. Eksp. Ter.* (6):70-73, 1974.

4141 THE EFFECT OF ADDITIONAL DAMAGE ON THE MITOTIC ACTIVITY OF THE RAT LIVER CELLS. *(Rus.)* Pukhaeva, R. G. (Tbilissi State Univ., USSR). *Exp. Morphol.* 76(3):721-724, 1974.

4142 CHANGES IN THE HISTONE CONTENT AND THE RATIO OF EASILY EXTRACTABLE AND FIRMLY BOUND HISTONE FRACTIONS IN CELL NUCLEI OF THE REGENERATING RAT LIVER. *(Rus.)* Ganelina, L. S.; Ovchinnikova, L. P. (Inst. Cytol., Leningrad, USSR). *Tsitologiia* 16(5):615-620, 1974.

4143 FINE STRUCTURE OF NUCLEI OF RAT HEPATOCYTES DURING REGENERATION AFTER PARTIAL HEPATECTOMY. *(Rus.)* Chelidze, P. V. (N. A. Natishvili Inst. Exp. Morphol., Tbilissi, USSR). *Biull. Akad. Nauk Gruzinskoi SSR* 76(3):717-721, 1974.

4144 STRESS-INDUCED BLOCKING OF THE MITOTIC CYCLE PROCESSES IN PROLIFERATING MURINE HEPATOCYTES. *(Rus.)* Uryvaeva, P. V.; Faktor, V. M. (Inst. Develop. Biol., Moscow, USSR). *Tsitologiia* 16(9):1100-1105, 1974.

4145 MORPHOFUNCTIONAL CHANGES OF RABBIT LIVER CELLS IN HEMORRHAGE. *(Rus.)* Mosevitch, T. N.; Korostovtseva, N. V.; Krivosheenko, T. N. (Inst. Cytol., Leningrad, USSR). *Tsitologiia* 16(10): 1214-1221, 1974.

4146 THE ABSORPTIVE FEATURES OF CELLS IN THE REGENERATING LIVER. *(Rus.)* Isanin, N. A.; Kuznetsova, G. B.; Yakovlev, A. Y. (Lab. Cell Physiol., Leningrad Univ., USSR). *Tsitologiia* 16(11): 1420-1423, 1974.

4147 THE INVESTIGATION OF THE PROTEIN CONTENT IN THE NUCLEI AND MITOCHONDRIA OF REGENERATING MOUSE LIVER. *(Rus.)* Revitch, G. G.; Mordukovitch, K. I.; Savina, M. I. (2nd Moscow Med. Inst., USSR). *Tsitologiia* 16(11):1382-1387, 1974.

4148 CHARACTERIZATION OF MICROSOMAL ELECTRON TRANSPORT COMPONENTS FROM CONTROL, PHENOBARBITAL, AND 3-METHYLCHOLANTHRENE TREATED MICE: I. DISTRIBUTION OF ELECTRON TRANSPORT COMPONENTS IN AMMONIUM-SULFATE FRACTIONS FROM MOUSE LIVER MICROSOMES. *(E.)* Mull, R. H.; Voigt, T.; Fleming, K. (Inst. Biophys. Radiobiol., Univ. Freiburg, Germany). *Biochem. Biophys. Res. Commun.* 64(3):1098-1106, 1975.

See also, 3787, 3789, 3802, 3882, 3985, 3986, 3987, 4180, 4539.

SECRETION AND METABOLISM

Intestine

4149 ENZYMES OF FRUCTOSE METABOLISM IN HUMAN SMALL INTESTINE MUCOSA. *(E.)* Heinz, F.; Schlegel, F.; Krause, P. H. (Med. Coll. Hannover, Germany). *Enzyme* 19(2):93-101, 1975.

The enzyme activities involved in fructose metabolism were measured in human intestine mucosa obtained 2 hr post mortem. Mucosa of the following gut sections were used: duodenum, jejunum, jejunum in the region of the flexura duodenojejunalis, distal jejunum, ileum middle region and ileum in the region of the valvula ileo cecalis. Only samples which showed a normal histological picture, where autolyzed cells were absent and no pathological changes could be observed, were chosen. Ketohexokinase, aldolase, alcohol dehydrogenases NAD- and NADP-dependent were found in all gut sections. The activity of aldehyde dehydrogenase was low in all sections tested. Triokinase could be found only in the duodenum and jejunum region and was absent in the ileum. Glycerate kinase was not present in the human intestine mucosa. Thus, the activities of all the enzymes involved in fructose metabolism were found in human small intestine mucosa, except D-glycerate kinase. There was, however, a broad standard deviation among the results, which may be due to differences in the nutritional state of the donors.

4150 EFFECT OF FAT FEEDING ON COMPLEX LIPID SYNTHESIS IN HAMSTER INTESTINE. *(E.)* Mansbach, II, C. M. (VA Hosp., Durham, N.C.). *Gastroenterology* 68(4):708-714, 1975.

Three enzymes engaged in complex lipid synthesis, diglyceride acyltransferase, cholinephosphotransferase, and lysolecithin acyltransferase, were studied in nonfasting hamster intestinal mucosa to determine if these enzymes were responsive to dietary fat supplementation. Male golden Syrian hamsters eating a fat-supplemented (corn oil) diet were compared to those eating a low fat (chow) diet. The diets were offered *ad libitum* for 1 week prior to death. The chow-fed group had gained an average of 4 g body weight and the fat-supplemented group, 5 g. The intestine was divided into four segments, pylorus to cecum, and the mucosa of each segment was scraped to yield a villous tip and crypt preparation. In the villous tips, fat supplementation had no effect on diglyceride acyltransferase and lysolecithin acyltransferase specific activity in the most proximal intestine. Both enzymes demonstrated increased activity in villous tips in response to fat feeding in the distal 3/4 of the gut. Cholinephosphotransferase activity was increased by fat feeding throughout the intestine in villous tips. Diglyceride acyltransferase and lysolecithin acyltransferase activity was greater in villous tips as compared to crypts. Cholinephosphotransferase activity was the same in crypts as in villous tips. Lysolecithin transferase was more responsive to fat feeding than was cholinephosphotransferase. It is claimed that these data lend support to the thesis that the lysolecithin acyltransferase pathway provides the lecithin associated with intestinal lipoprotein formation. Of four microsomal enzymes not related to lipid metabolism, only one, NADPH-cytochrome c reductase, was affected by fat feeding suggesting that the microsomal enzyme response to fat feeding was relatively specific.

4151 THE EFFECT OF ACUTE ETHANOL TREATMENT ON LIPID-REESTERIFYING ENZYMES OF THE RAT SMALL BOWEL. *(E.)* Rodgers, J. B.; O'Brien, R. J. (Albany Med. Coll., N.Y.). *Am. J. Dig. Dis.* 20(4): 354-358, 1975.

The acute effects of intraduodenal administration of ethanol (5 g/kg) on intestinal activities of lipid-reesterifying and disaccharidase enzymes of the small bowel were studied in male Sprague-Dawley rats. Animals were infused intraduodenally with ethanol or isocaloric glucose for 5 hr at 2.4 ml/hr. Animals were fasted overnight, sacrificed, and the small intestine distal to the ligament of Treitz removed and divided into the jejunal and ileal segments. Mucosal samples of each segment were scraped, homogenized, and assayed for enzyme activity. Acyl-coenzyme A(CoA):monoglyceride acyltransferase, acyl-CoA synthetase (acid:CoA ligase), sucrase, and lactase assays were performed on jejunal samples; acyl-CoA synthetase assay was performed on ileal samples. Ethanol administration produced greater activities of the lipid-reesterifying enzymes in the jejunum than did glucose administration. Ileal specific activity of acyl-CoA synthetase was also increased in the ethanol group. No effect of ethanol on jejunal disaccharadase enzyme activities was seen. It is concluded that ethanol, given acutely, has a specific stimulating effect on intestinal enzymes involved in lipid absorption.

4152 ILEAL HCO_3 SECRETION: RELATIONSHIP TO Na AND Cl TRANSPORT AND EFFECT OF THEOPHYL-

LINE. *(E.)* Sheerin, H. E.; Field, M. (Harvard Med. Sch., Boston, Mass.). *Am. J. Physiol.* 228(4):1065-1074, 1975.

Interrelationships among Na, Cl, and HCO_3 transport processes were examined in short-circuited New Zealand white, male rabbit ileal mucosa. Na and Cl fluxes at different serosal HCO_3 concentrations were determined either in paired experiments on tissues from the same animal or in nonpaired but randomized experiments. As serosal HCO_3 concentration was increased from 10 to 50 mM (pH from 7.1 to 7.8), net Na absorption decreased from 4.6 to 0.3 $\mu Eq/hr/cm^2$, net Cl flux changed from absorption of 0.9 to secretion of 0.9 and a net HCO_3 secretion of 3.0 developed. A similar change in net Cl flux was also observed when serosal CO_2 was altered at constant HCO_3 concentration. In Cl-free SO_4-Ringer, serosal alkalinization produced net HCO_3 secretion which was not significantly less than that observed in Cl-containing Ringer. Theophylline caused secretory changes in net Na and Cl fluxes at both 10 and 50 mM serosal HCO_3 concentrations. Theophylline did not alter net HCO_3 flux in Cl-Ringer but increased net HCO_3 flux in SO_4-Ringer. Total direct current conductance was decreased by both serosal alkalinization and theophylline. Short-circuit current was consistently increased by theophylline but not by serosal alkalinization. These results indicate that ileal ion transport is regulated in part by serosal pH and/or HCO_3 concentration and that resulting changes in Cl and HCO_3 transport are coupled 1:1 with changes in Na transport. It is also concluded that HCO_3 secretion does not require the presence of Cl in the bathing medium.

4153 DIGESTION AND ABSORPTION OF THE CONSTITUENTS OF CONCENTRATED MILK PRODUCTS WITH SPECIAL REFERENCE TO THE EFFECT OF INCREASED QUANTITIES OF LACTOSE. *(Pol.)* Cieslakowa, D.; Palaszewska, M.; Ziemlanski, S. (Inst. Nutr. Food Prod., Warsaw, Poland). *Przegl. Lek.* 31(2):328-333, 1974.

A total of 420 male Wistar rats (6 and 10 weeks old) were fed diets containing various amounts of lactose (from 3 to 15%). The following parameters were studied: digestion of protein and fat; the rate at which protein, fat, and lactose passed into the duodenum; the absorption of these nutrients in small intestine; and concentrations of α-amino nitrogen, fat, and glucose in the portal and cardiac blood. With increasing amounts of dietary lactose the rate of gastric emptying decreased, especially during the first 30 minutes. Increasing dietary lactose from 3% to 12% had no effect on digestion and absorption of nitrogen compounds from the small intestine, but 15% lactose slightly decreased protein digestion and decreased digestion and absorption of fat. Increasing dietary lactose from 3% to 15% caused a corresponding increase in rate at which liberated glucose was digested and absorbed.

4154 ACUTE AND CHRONIC EFFECTS OF ETHANOL ON INTESTINAL LIPID METABOLISM. *(E.)* Baraona, E.; Pirola, R. C.; Lieber, C. S. (VA Hosp., Bronx, N. Y.). *Biochim. Biophys. Acta* 388(1):19-28, 1975.

The effects of both the addition *in vitro* of various ethanol concentrations and the previous *in vivo* administration of ethanol on the ability of intestinal slices to oxidize fatty acids and to synthesize triacylglycerols were assessed in CD male rats. The rats were pair-fed a synthetic liquids diet containing 18% of the total calories as protein, 35% as fat, 11% as carbohydrate, and 36% either as ethanol or additional carbohydrate (control diet) for 3-4 weeks. Animals were sacrificed and the small intestine, from the angle of Treitz to the ileo-cecal junction, was stripped from the mesentery. Segments of the intestine from the proximal 20 cm of jejunum and from the ileum (between 30 and 10 cm proximal to the cecum) either were sliced in rings for the metabolic studies *in vitro* or were used for the enzyme assays. The addition of ethanol to the incubation medium in concentrations likely to be achieved in the upper jejunum after moderate drinking inhibited both palmitate and acetate oxidation, CO_2 production and triacylglycerol synthesis, but enhanced the esterification of fatty acid with ethanol. The concentrations required for the inhibitory effect were much higher than those needed to saturate enzyme systems known to participate in ethanol oxidation. *In vivo* administration of ethanol-containing diets produced persistent changes of the intestinal slices with respect to fatty acid oxidation and triacylglycerol synthesis. Acute intragastric administration of ethanol (3 g/kg) 1 hr prior to sacrifice, inhibited both processes in slices obtained from the jejunum, but not in those derived from the ileum. Chronic ethalol feeding increased the ability for fatty acid oxidation and triacylglycerol synthesis both in the jejunum and in the ileum. This stimulatory effect was associated with significant enhancement of palmitoyl-coenzyme A synthetase activity, suggesting increased fatty acid activation. These findings suggest that the high concentrations of ethanol to which the small intestine is exposed during drinking inhibit fatty acid oxidation and esterification to triacylglycerols apparently as a consequence of a direct damaging effect of alcohol. It is further suggested that prolonged ethanol administration promotes functional and structural changes which result in an increased capacity for fatty acid oxidation and triacylglycerol synthesis.

4155 INFLUENCE OF SECRETIN AND PENTAGASTRIN ON THE CIRCADIAN RHYTHM OF CELL PROLIFERATION IN THE INTESTINAL MUCOSA IN RATS. *(E.)* Pansu, D.; Berard, A.; Dechelette, M. A.; Lambert, R. (INSERM, Edouard Herriot Hosp., Lyon, France). *Digestion* 11 (3/4):266-274, 1974.

The effects of pentagastrin and secretin on DNA synthesis in rat jejunal mucosa were studied. Wistar rats were fasted 24 hr and given 25 μg/100 g pentagastrin or 3.5 U/100 g secretin, and sacrificed after 0, 4, 8, 12, 16, 20, and 24 hr. Controls were given saline. For the pentagastrin 6 rats were paired with 6 controls; for secretin, 7 rats paired with 6 separate controls. For DNA labeling, rats were injected i. p. with 70 Ci/100 g [^{3}H] thymidine 30 min before killing. Histoautoradiography was done on sections of intestine 10 cm distal to the ligament of Treitz. Labeling index was the per-

centage of labeled nuclei counted in the proliferative compartment (the first 30 cells in the crypt) and mitotic index was the percentage of nuclei in the mitotic state determined for 1800 cells/crypt. After pentagastrin injection, labeling index was 31.73 at time 0, rose to 46.7 at 12 hr, and stabilized at this level until 24 hr later. In contrast, controls had a labeling index of 31.73 at time 0 which rose to 45.0 at 12 hr and returned to 36.45 at 24 hr. Treated cells had significantly higher indices than controls at 20 and 24 hr. The increased number of labeled cells in comparison to controls was in the upper part of the crypt. The mitotic index after injection rose from 7.31 at time 0 to 10.26, 24 hr later, similar to the control values of 7.30 at time 0 and 10.36 after 24 hr. After secretin injection the nocturnal increase in labeling index disappeared, there being no significant difference between values at time 0 (33.25) and 24 hr (38.10). The mitotic index was 7.83 at time 0 and remained steady, thus becoming significantly lower than control values after 24 hr (10.52). The decreased number of labeled cells in comparison to the controls was in the lower part of the crypt. It is concluded that pentagastrin has a stimulating effect on the circadian rhythm of cell renewal, increasing the labeling index and increasing the proliferative zone, while secretin has an inhibitory effect, decreasing both labeling and mitotic indices.

4156 CIRCADIAN VARIATIONS IN THE REACTION OF THE EPITHELIAL CELLS OF THE INTESTINE AND CORNEA OF MICE IN RESPONSE TO THE ACTION OF COLCHICINE. *(Rus.)* Semenova, N. F. (Khabarovsk Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 78(12):68-71, 1974.

4157 INTESTINAL DISACCHARIDES IN RATS TREATED WITH 5-FLUOROURACIL. *(Ger.)* Hartwich, G.; Domschke, W.; Matzkies, F.; Pesch, H. J.; Prestele, H. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Klin. Wochenschr.* 52(19):930-938, 1974.

4158 CHOLINESTERASE AND MONOAMINOXIDASE ACTIVITY OF THE SMALL INTESTINAL MUCOSA DURING PROTRACTED ACTIVITY OF ITS RESORPTION MECHANISM. *(Ukr.)* Iaremko, E. O. (Lvov Med. Inst., USSR). *Fiziol. Zh.* 21(1):52-56, 1975.

4159 PHOSPHOPYRIDOXAL COMPLEXES WITH HISTAMINE AND HISTIDINE: (3) THE INFLUENCE OF PRESUMED COMPLEX ON ACTIVITY OF RAT INTESTINAL HISTAMINASE. *(E.)* Sasiak, K.; Kierska, D.; Boguslawski, M.; Maslinski, C. (Dep. Biog. Amines, Polish Acad. Sci., Lodz). *Agents Actions* 5(1):25-30, 1975.

4160 EXPERIMENTAL RESEARCH ON SURGICAL TREATMENT OF OBESITY: INFLUENCE OF JEJUNOILEAL BYPASS WITH LIPECTOMY AND OF LIPECTOMY ALONE ON SOME PARAMETERS OF GROWING ANIMALS. *(It.)* Petronio, R.; Nicoli, N.; Giovaninetti, G. D.; Delaini, E. T. (Clin. Gen. Surg, Surg. Ther., Univ. Padua, Italy). *Chir. Ital.* 26(3):185-194, 1974.

4161 REGIONAL VARIATION IN GLYCOLYTIC ENZYME ADAPTATION TO DIETARY SUGARS IN RAT SMALL INTESTINE. *(E.)* Espinoza, J.; Hritz, A.; Kaplan, R.; Clark, S. B.; Rosenweig, N. S. (St. Luke's Hosp. Cent., Columbia Univ., New York, N.Y.). *Am. J. Clin. Nutr.* 28(5): 453-458, 1975.

4162 THE EFFECTS OF MALTITOL ON RAT INTESTINAL DISACCHARIDASES. *(E.)* Yoshizawa, S.; Moriuchi, S.; Hosoya, N. (Fac. Med., Univ. Tokyo, Japan). *J. Nutr. Sci. Vitaminol.* 21(1):31-37, 1975.

4163 MOLECULAR ACTION OF VITAMIN D IN THE CHICK INTESTINE. *(E.)* Lawson, D. E. M.; Emtage, J. S. (Dunn Nutr. Lab., Univ. Cambridge, England). In: *Vitamins and Hormones. Advances in Research and Applications.* edited by Harris, R. S. (New York: Academic Press, 1974).

See also, 3792, 3801, 3802, 3840, 4177, 4185.

GENERAL

4164 EFFECTS OF 400 R WHOLE-BODY X-IRRADIATION ON 5-HYDROXYTRYPTAMINE CONTENT OF THE RAT GASTROINTESTINAL TRACT. *(E.)* Penttila, A.; Kormano, M.; Ahonen, A. (Dep. Forensic Med., Univ. Helsinki, Finland). *Strahlentherapie* 149(4):426-437, 1975.

The effects of a single 400 r whole-body dose of x-irradiation on the regional 5-hydroxytryptamine content of the gastrointestinal tract of adult and young male Sprague-Dawley rats were studied from 4 hr to 40 days after irradiation using comparative chemical and morphological methods. The 5-hydroxytryptamine content of tissue pieces was determined in µg/whole organ, and per wet weight of tissue as well as per tissue protein using spectrophotofluorometry. After a single dose of radiation the total 5-hydroxytryptamine content of the stomach and intestinal tract was significantly lower in the irradiated than in the control rats 4 hr and 1 and 2 days after irradiation. The total amine content of the gastrointestinal tract returned to normal 5 days after irradiation and remained constant thereafter. Some

differences were found between the various regions. Some minor morphological changes were seen in enterochromaffin cells 1 and 2 days after irradiation but not later. The effects of irradiation of the gastrointestinal tissues were stronger in young rats, in which no complete recovery of 5-hydroxytryptamine occurred within 45 days. The results support the view that irradiation exerts a direct damaging effect on the gastrointestinal 5-hydroxytryptamine storage, which is associated with liberation of 5-hydroxytryptamine from amine-containing cells. It is concluded that the duration of the effect varies with age and intestinal segment concerned and that the maturation of the animals affords some protection against its effects.

4165 MICROCIRCULATION OF THE NORMAL AND INFLAMED CANINE PANCREAS. *(E.)* Schiller, W. R.; Anderson, M. C. (Med. Coll. Ohio, Toledo). *Ann. Surg.* 181(4):466-470, 1975.

The microcirculation of the pancreas of five normal dogs and the inflamed gland of experimental pancreatitis induced in another five dogs was studied using an intraarterial injection of Microfil, a silicon rubber compound with a particle size of 1-3 μ. Laparotomy was performed through a midline incision upon 10 anesthetized adult mongrel dogs. In the five control animals, Microfil was injected into the cranial pancreaticoduodenal artery. The pancreas and adjacent duodenum were immediately excised between clamps and stored in a refrigerator overnight to allow polymerization of the Microfil. In the other five, pancreatitis was induced by a transduodenal retrograde injection of 150,000 U trypsin in 15 ml saline into the major pancreatic duct. The animals were re-operated 24 hr later, Microfil injected according to the same procedure as for the 3 controls, and specimens obtained and processed. Specimens were examined with a dissecting microscope and photographed. Duodenal vasculature and lobular vessels of the pancreas were studied as were those supplying the duct. Interlobular vessels were well visualized in the normal pancreas and intralobular vessels formed a fine reticular pattern throughout the cleared lobules. A complex network of vessels was observed in the pancreatic duct, apparently derived from the interlobular vessels. Intraductal trypsin injection produced focal areas of pancreatitis associated with edema, poor vascular filling and spastic changes of the lobular vessels. Extravasation of Microfil, although not apparent on normal specimens, was prominent in inflamed specimens and suggested vascular weakness and disruption. Pancreatic lobules adjacent to inflammatory areas showed definite evidence of dilatation. The inflamed pancreatic ducts were markedly edematous, thickened, and showed incomplete vascular filling. When the duodenum adjacent to the pancreatitis was injected with Microfil, edema and vasoconstriction were especially prominent in the duodenal muscular layer. It is suggested that the compromise of parenchymal circulation in combination with enzyme extravasation into the gland parenchyma may contribute to increased severity of the pancreatic inflammation; a re-distribution of pancreatic blood flow occurred with hyperperfusion of minimally involved areas and stasis in the inflamed pancreas. The changes in the duodenal microcirculation suggest that the abnormal gastrointestinal motility associated with pancreatitis may result in part from an altered circulation in the duodenal muscularis.

4166 THE EFFECT OF ELECTRICAL STIMULATION OF THE CELIAC GANGLION ON PANCREATIC BLOOD FLOW. *(Fr.)* Varga, B.; Folly, G.; Papp, M. (Inst. Exp. Med., Budapest, Hungary). *Lyon Chir.* 70(3): 168-170, 1974.

Male dogs averaging 16 kg were subjected to electrical stimulation of the left celiac ganglion with 2 min bursts of rectangular electrical impulses (20 impulses/sec at 2.5, 5, and 10 V). The pancreatic blood flow was measured by heated thermocouples and recorded on a fluorograph, while the arterial pressure was measured in a femoral artery. The stimulus amplitude was varied in a random sequence, and the corresponding blood flow was recorded. Electrical stimulation raised the blood pressure and decreased the pancreatic blood flow in proportion to the amplitude of the stimulus. When the pancreatic blood flow was increased by injecting dehydrocholic acid (4 mg/Kg/min i.v.), it dropped even more significantly when an electrical impulse was applied. It is suggested that during acute pancreatitis the reflex contraction of pancreatic blood vessels aggravates the disease. When laparatomy is performed, the celiac ganglion should be amply infiltrated with a local anesthetic to prevent spastic contraction of the pancreatic vessels.

4167 BACTERIAL DEGRADATION OF β-MURICHOLIC ACID IN THE RAT. *(Fr.)* van Heijenoort, Y.; Sacquet, E.; Riottot, M. (Lab. Germfree Anim., CNRS, Gif-sur-Yvette, France). *Nutr. Metab.* 17(2):65-73, 1974.

Bacterial degradation of β-muricholic acid was studied by thin layer chromatographic analysis of the feces of two holoxenic male Fischer rats, aged 3 months, which had been injected in the stomach with an excess of β-muricholic-4-^{14}C acid (12.25 mg, containing 0.44 μCi). By the second day after injection, almost 95% of the radioactivity had been excreted and was distributed among various degradation products of β-muricholic acid, principally hyodeoxycholate. When the hyodeoxycholate zone of the chromatograms was eluted and rechromatographed, 91% of the radioactivity originally injected was found at the level of the control methyl hyodeoxycholate. The remaining radioactivity was distributed at the levels of two unknown substances, four methyl esters of acids, particularly 3α, 6β-dihydroxy-5β-methyl cholanoate, and the chromic oxidation product of methyl β-muricholate. *In vitro* incubation of 5 mg β-muricholic acid-4-^{14}C with a homogenate of rat cecum resulted in degradation of only 22% of the β-muricholic acid. The radioactivity appeared on the chromatograms mainly at the level of an unknown substance and at the levels of two methyl cholanoates. There was no significant radioactivity at the levels of hyodeoxycholate and 3α, 6β-dihydroxy-5β-methyl cholanoate. These results contrast with observations on axenic

rats in which β-muricholic acid is the main bile acid. The degradation pathway of β-muricholic acid by intestinal flora remains obscure.

4168 ARTERIOVENOUS INTESTINAL COMMUNICATIONS IN SHOCK. EXPERIMENTAL STUDY USING TAGGED MICROSPHERES. *(E.)* Androulakis, J.; Androulakis, G.; Vezeridis, M.; Kyriakou, K. (Aretaeion Hosp., Athens, Greece). *Int. Surg.* 60(4):206-208, 1975.

Polystyrene microspheres, 25 μ in diameter labeled with ^{141}Ce and ^{85}Sr, were used to study the influence of oligemic shock on arteriovenous anastomoses of the small intestine mucosa of five dogs and five white New Zealand rabbits. One ml of [^{141}Ce] microsphere solution was injected into a catheter in the superior mesenteric artery. The intraaortic pressure was recorded through a catheter connected to an electromanometer. Oligemic shock was created by gradual peripheral arterial bleeding in 60-100 min, until blood pressure dropped to 20-30 mm Hg, and was maintained at this level for at least 30 min. At this stage, the ^{85}Sr solution was injected. Following injection of the second microsphere solution, the animal was sacrificed, using an i.v. dose of KCl. Radioactivity was measured in a specimen of small bowel corresponding to the injected branch of the superior mesenteric artery and one from the liver, as well as in the syringes before and after microsphere injection. No radioactivity was detected in the liver either before or during oligemic shock. Most of the radioactivity was retained in the intestinal mucosa. These data suggest that the microspheres did not pass through the microcirculation of the intestine, indicating that there are no arteriovenous anastomoses in the intestinal mucosa, or that if they do exist they are less than 25 μ in diameter. The last possibility is considered the more probable since there are many hemodynamic and metabolic indications for the existence of shunts and their opening during shock.

4169 THE NORMAL COLONIC BACTERIAL FLORA. *(E.)* Hill, M. J.; Drasar, B. S. (Colindale Hosp., London, England). *Gut* 16(4):318-323, 1975.

4170 THE MULTIPLICATION OF Trypanosoma equiperdum IN A DIFFUSION CHAMBER IMPLANTED IN MICE. *(Fr.)* Pautrizel, A. N.; Mattern, P.; Pautrizel, R. (Immunol. Parasit. Biol. Lab., Univ. Bordeaux II, France). *C. R. Acad. Sci. [D] (Paris)* 280 (15):1825-1828, 1975.

4171 RESEARCH ON THE DEVELOPMENT OF A MICRO-ORGANISM-FREE SMALL INTESTINE IN ADULT MICE. *(Ger.)* Meyer, E.; Stelzner, A.; Koch, H.; Kittlick, M. (Inst. Med. Microbiol., Friedrich Schiller Univ., Jena, Germany). *Wiss. Z. Friedrich Schiller Univ. Jena Math. Naturwiss. Reihe* 23(3/4):497-502, 1974.

4172 PLEOMORPHIC VIRUS-LIKE PATRICLES IN HUMAN FAECES. *(E.)* Mathan, M.; Swaminathan, S. P.; Mathan, V. I.; Yesudoss, S.; Baker, S. J. (Christian Med. Coll. Hosp., Vellore, India). *Lancet* 1(7915):1068-1069, 1975.

4173 FRACTIONATION OF RNA FROM THE LIVER, HEPATOMAS, AND GASTRIC MUCOSA BY PREPARATIVE POLYACRYLAMIDE GEL ELECTROPHORESIS. *(Rus.)* Kalinovskii, V. P.; Goriukhina, T. A.; Seits, I. F. (N. N. Petrov. Res. Inst. Oncol., Leningrad, USSR). *Vopr. Med. Khim.* 20(5):540-544, 1974.

4174 PRESENT STATUS OF THE d-UROBILINS. *(E.)* Petryka, Z. J.; Weimer, M.; Lightner, D. A.; Chedekel, M.; Bovey, F. A.; Moscowitz, A.; Watson, C. J. (Northwest. Hosp., Minneapolis, Minn.). *Ann. NY Acad. Sci.* 244:521-532,1975.

4175 THE NEURONAL POPULATION OF THE INTRAMURAL GANGLIA IN THE GASTROINTESTINAL TRACT. *(Rus.)* Krokhina, E. I.; Chuvilskaia, L. M. (Inst. Norm. Pathol. Physiol., Moscow, USSR). *Biull. Eksp. Biol. Med.* 79(3):110-113, 1975.

4176 THE INGESTION OF ASBESTOS FIBERS. *(E.)* Webster, I. (South African Med. Res. Counc., Johannesburg). *Environ. Health Perspect.* 9(12):199-202, 1974.

4177 THE EFFECT OF RETROANASTOMOSIS OF PANCREATIC CROSS-SECTION WITH SMALL INTESTINE ON HEALTHY PANCREATIC PARENCHYMA IN DOGS. *(Pol.)* Taczanowski, W. (Surg. Clin. II, Warzaw, Poland). *Pol. Przegl. Chir.* 46(12):1645-1650, 1974.

4178 DEMONSTRATION OF A CARCINOFETAL ANTIGEN ON THE MEMBRANE OF INTESTINAL CARCINOMA CELLS IN THE RAT. *(Fr.)* Martin, F.; Martin, M.; Knobel, S.; Bordes, M. (no affil.). *Ann. Immunol. (Paris)* 126 C(1):100, 1975.

4179 DIURNAL VARIATIONS IN RADIOSENSITIVITY OF MOUSE INTESTINE. *(E.)* Hendry, J. H. (Christie Hosp., Withington, England). *Br. J. Radiol.* 48(568):312-314, 1975.

4180 ASEPTIC ENDOTOXEMIA IN RADIATION INJURY AND GRAFT-VS-HOST DISEASE. *(E.)* Walker, R. I.; Ledney, G. D.; Galley, C. B. (Armed Forces Radiobiol. Res. Inst., Bethesda, Md.). *Radiat. Res.* 62(2):242-249, 1975.

4181 INTERACTIONS OF BILIRUBIN AND OTHER LIGANDS WITH LIGANDIN. *(E.)* Kamisaka, K.; Listowsky, I.; Gatmaitan, Z.; Arias, I. M. (Albert Einstein Coll. Med., Yeshiva Univ., Bronx, N.Y.). *Biochemistry* 14(10):2175-2180, 1975.

4182 A PROFILE OF THE RAT GASTROINTESTINAL TOXICITY OF DRUGS USED TO TREAT INFLAMMATORY DISEASES. *(E.)* Shriver, D. A.; White, C. B.; Sandor, A.; Rosenthale, M. E. (Wyeth Lab., Inc., Philadelphia, Pa.). *Toxicol. Appl. Pharmacol.* 32(1):73-83, 1975.

4183 ANIMAL MODEL FOR DETERMINING THE NO-EFFECT LEVEL OF ANTIMICROBIAL DRUG ON DRUG RESISTANCE IN THE LACTOSE-FERMENTING ENTERIC FLORA. *(E.)* Rollins, L. D.; Gaines, S. A.; Pocurull, D. W.; Mercer, H. D. (Food Drug Admin., Beltsville, Md.). *Antimicrob. Agents Chemother.* 7(5):661-665, 1975.

4184 EXPERIMENTAL CATHETER OBSTRUCTION OF THE GASTRIC CORONARY VEIN. POSSIBLE TECHNIQUE FOR PERCUTANEOUS INTRAVASCULAR TAMPONADE OF THE GASTROESOPHAGEAL VARICES. *(E.)* Rosch, J.; Goldman, M. L.; Dotter, C. T. (Univ. Oregon Med. Sch., Portland). *Invest. Radiol.* 10(3):206-211, 1975.

4185 THE EFFECT OF S-2-(3-AMINOPROPYLAMINO)ETHYLPHOSPHOROTHIOIC ACID (WR-2721) ON INTESTINAL CRYPT SURVIVAL. I. 4 MeV X-RAYS. *(E.)* Sigdestad, C. P.; Connor, A. M.; Scott, R. M. (Sch. Med., Univ. Louisville, Ky.). *Radiat. Res.* 62(2):267-276, 1975.

DIAGNOSTIC PROCEDURES

4186 PREOPERATIVE DIAGNOSIS OF THE MECKEL'S DIVERTICULUM BY SEQUENTIAL 99m TECHNETIUM PERTECHNETATE SCANNING. *(E.)* Lee, H. K.; Schwartz, I. S.; Nipper, T.; Gichon, G.; Beck, A. R. (City Hosp. Cent. Elmhurst, N.Y.). *Mt. Sinai J. Med NY* 42(2):136-141, 1975.

A 28-month-old female presented with a 2 hr history of passing bloody stools. There was no evidence of abdominal pain, nausea, or other symptoms, but there had been a 6 day episode of bloody stools at the age of 18 months. During the first 12 hr of observation no further bloody stools were passed. Twenty-four hr after admission an abdominal scan was performed. The stomach was emptied by nasogastric tube and 50 mg sodium iodide was injected i.v. to block background activity. The infant was placed supine under the scanning apparatus and 1.5 mCi of ^{99}m Tcpertechnetate was injected through a left leg vein. Sequential scintiphotos were taken for the following 4 3/4 hr. These demonstrated a round focal area of increased activity in the right mid-abdomen, simultaneously visualized with stomach activity. Meckel's diverticulum was diagnosed and confirmed on surgery. The diverticulum-bearing segment of ileum was resected, and the appendix removed. Histologic study showed normal ileal mucosa lining the segment, and numerous gastric parietal acid-secreting cells in the diverticulum that had caused extensive ulceration of the ileo-diverticular junction. Simultaneous scanning of the specimen showed a high concentration of technetium in the parietal cell portion. The appendix appeared normal. It is concluded that sequential scanning is useful in diagnosis of Meckel's diverticulum, and makes possible simultaneous visualization of the diverticulum and stomach activity.

4187 A NEW HYDROGEN ION TELEMETRY TECHNIQUE FOR EVALUATING GASTROESOPHAGEAL REFLUX. *(E.)* Brooks, J.; Kia, D.; Meguid, M.; Lewicki, A.; Membreno, A.; Culebras, J. (Peter Bent Brigham Hosp., Harvard Med. Sch., Boston, Mass.). *Surg. Gynecol. Obstet.* 140(5):749-955, 1975.

A new pH capsule telemetry technique was used to measure the pH fluxes in the upper part of the gastrointestinal tract in normal volunteers, symptomatic patients, and in those with hiatal herniorrgraphy during the preoperative and postoperative periods. The telemetry system consists of a pH-measuring radio capsule which measures the H^+ activity of the surrounding medium to ± 0.2 pH unit in a range of pH 1-8. The reference electrode is self-contained. The capsule is small (0.8 x 7.9 mm) and contains a radiotransmitter which emits a signal determined by the pH of the surrounding medium. This signal is converted to a linear graph on a strip chart recorder. The capsule was swallowed with ease and with minimal discomfort by a fasting patient. The mean basal pH range in the esophagus 5 cm above the gastroesophageal junction in fasting normal persons was 7.5. A significant drop occurred when the capsule entered the empty fasting stomach (3.8) and dropped further to 1.8 with the addition of 240 ml HCl. The mean basal esophageal pH in a group of patients with symptoms of reflux was 7.6. A significant, sharp and sudden pH drop to 2.6 occurred on advancing the capsule into the fasting stomach. On returning the capsule to a position 5 cm above the gastroesophageal junction, the pH stabilized to 7.0 which differed significantly from the basal esophageal pH in this group. In these patients, acid reflux into the lower part of the esophagus was consistently induced by this maneuver. In six patients with hiatal hernia, no reflux was noted after a transabdominal Nissen fundoplication or a Belsey-type buttress procedure. In two patients who underwent an Allison repair without fundioplication, reflux persisted postoperatively. It is concluded that this pH capsule telemetry is easy to perform on an ambulatory basis and allows an accurate and reproducible determination of the presence or absence of esophageal reflux in patients with and without hiatal hernia. Its correlation with symptomatic reflux is higher than that found with a conventional gastrointestinal series examination.

4188 OPERATIVE BIOPSY OF THE PANCREAS. *(E.)* George, P.; Brown, C.; Gilchrist, J. (R. Free. Hosp., London, England). *Br. J. Surg.* 62(4): 280-283, 1975.

A retrospective study was undertaken of 47 patients (19-84 yr) who had open pancreatic biopsies during the course of a laparotomy to determine whether the procedure was safe and whether worthwhile information was obtained. The most common indication for biopsy (35 cases) was histological confirmation of a suspected carcinoma. Pancreatic biopsies were also taken from patients in whom the nature of the pancreatic pathology was in doubt and from patients who had preoperative investigations suggesting pancreatic disease but who had a macroscopically normal pancreas at operation. The anterior surface of the pancreas was exposed by opening the lesser sac. The biopsy was taken from the most superficial and firmest part of the lesion using a tenotomy knife; a small pyramid (0.3 x 0.3 x 0.3 cm) of tissue was removed. The exposed raw area was covered with either a piece of Oxycel or omentum, lightly held in place by two chromic catgut sutures. In this series of patients none of the postoperative complications could be attributed to the biopsy. It is claimed that the procedure is safe using the technique described provided that the biopsy is kept as superficial as possible and care is taken not to biopsy the region of the main pancreatic duct. It is concluded that the pancreatic biopsy is of value. If carcinoma is found, then there is unequivocal proof of the lesion. If chronic pancreatitis or a normal pancreas is found, then there is contributory evidence of a non-malignant condition. It is suggested that in the vast majority of cases (91% in this group) reliable information will be obtained.

4189 EMERGENCY PERITONEOSCOPY. *(Fr.)* Rachail, M.; Coralle, J.; Maurel, G. (Univ. Hosp. Cent. Grenoble, France). *Arch. Fr. Mal. App. Dig.* 63(2):103-108, 1974.

Emergency peritoneoscopies were performed on 80 pa-

tients: 14 with abdominal trauma (of the diaphragm, spleen, liver, pancreas, abdominal wall) or hemoperitoneum and 66 with acute abdominal diseases (cholecystitis, pancreatitis, cholangitis, appendicitis, intestinal obstruction, malignant tumors, tuberculosis, sigmoiditis). The procedure was well tolerated in all but 1 patient whose small intestine was punctured, and led to a correct diagnoses in 71 of 80 cases. It is recommended as a safe and efficient diagnostic procedure in cases of acute abdomen, even in comatose patients.

4190 THE TREHALOSE TOLERANCE TEST IN THE INVESTIGATION OF INTESTINAL ABSORPTION IN PATIENTS ON INTERMITTENT HEMODIALYSIS. *(Ger.)* Pointner, H.; Flegel, U.; Bayer, P. M.; Schmidt, P. (1st Med. Clin., Univ. Vienna, Austria). *Wien. Klin. Wochenschr.* 86(22):691-693, 1974.

The trehalose test for intestinal malabsorption was performed on 9 uremic patients receiving intermittent hemodialysis. None had any signs of malabsorption. They were given 50 g α-D-trehalose one day before their weekly dialysis. One week later they received the same amount of glucose. The blood sugar content was checked at regular intervals from 0 to 120 minutes after each test, and a trehalose absorption index (TAI) was computed from these data. The same procedure was applied to a group of 5 normal patients. No significant difference in the TAI was found between the 2 groups. Thus the trehalose tolerance test is valuable in the diagnosis of malabsorption in patients with renal insufficiency.

4191 A SIMPLE METHOD FOR CALCULATING HEPATIC BILIRUBIN CLEARANCE: ITS APPLICATION IN PATIENTS WITH GILBERT'S SYNDROME. *(Ger.)* Kutz, K.; Egger, G.; Bachofen, H.; Preisig, R. (Inst. Clin. Pharmacol., Univ. Bern, Switzerland). *Helv. Med. Acta* 37(5/6):386-387, 1974.

A simple method for the calculation of the hepatic bilirubin clearance in the diagnosis of Gilbert's syndrome is described. The bilirubin production in mg/day is calculated as the product of blood volume, hemoglobin concentration, daily rate of hemolysis, equalling 100/age of erythrocytes, and a factor 36.3. The age of the erythrocytes was determined by the ^{51}Cr method. This method, also requiring the determination of the serum bilirubin concentration, permits differentiation between patients with Gilbert's syndrome and normal subjects, as well as patients with increased hemolysis.

4192 IN VIVO STAINING OF GASTRIC MUCOSA: ITS APPLICATION TO ENDOSCOPIC DIAGNOSIS OF INTESTINAL METAPLASIA. *(E.)* Ida, K.; Hashimoto, Y.; Kawai, K. (Murakami Mem. Hosp., Gifu, Japan). *Endoscopy* 7(1):18-24, 1975.

In vivo staining of the human gastric mucosa was examined using the indirect dye scattering method with p.o. methylene blue. Endoscopic examination following methylene blue administration revealed complete staining of the gastric mucosa within 2-3 min. Postgastrectomy examination of dye-stained specimens indicated that the dye was taken up by the surface epithelial layer of the gastric mucosa. The stained region was generally located in the pyloric gland area, and areas of intestinal metaplasia were selectively stained when present. Staining was observed in 105 (65.6%) of 160 patients, with staining frequency by disease as follows: gastric polyp, 93%; gastric ulcer, 55%; gastric ulcer scar, 77%; gastric carcinoma, 50%. These values increased with age. Ninety percent of the stained region was of the antral type, the antral region being primarily stained; the remaining 10% was of the intermediate type in which the staining ranged as far as the upper boundary of the pyloric gland area. The most common staining pattern (83%) was one in which granules were stained; in the remaining cases, small depressed regions between the granules were stained. The results suggest that granular-antral staining is the most commonly observed pattern. This *in vivo* staining method offers promise as a means of improving the endoscopic diagnosis of intestinal metaplasia, gastric cancer, and benign atypical epithelium.

4193 EVALUATION OF WHOLE AND PURIFIED HYDATID FLUID ANTIGENS IN THE DIAGNOSIS OF HUMAN HYDATIDOSIS BY THE IMMUNOELECTROPHORESIS TEST. *(E.)* Varela-Diaz, V. M.; Guisantes, J. A.; Ricardes, M. I.; Yarzabal, L. A.; Coltorti, E. A. (Pan Am. Zoonoses Cent., World Health Org., Ramos Mejia, Argentina). *Am. J. Trop. Med. Hyg.* 24(2):298-303, 1975.

The efficacy of whole sheep hydatid cyst fluid (WHF) and purified hydatid fluid antigen (PHF) in the diagnosis of human hydatid disease was evaluated with the immunoelectrophoresis (IEP) test. Both antigens were tested with preoperative and postoperative sera from 75 patients with hyatid disease, sera from 41 patients with other parasites, sera from 41 patients with nonparasitic diseases, and sera from 20 healthy donors. Among the hydatid sera, 86.7% yielded positive results with the PHF antigens, 70.7% yielded positive results with the WHF antigen, and 13.3% yielded negative results with both antigens. None of the non-hydatid sera reacted positively with the WHF antigen, but 28% of the sera from patients with other parasitic and non-parasitic diseases yielded positive results with the purified antigen. Among preoperative hydatid patients, the *Echinococcus granulosus*-specific arc 5 criterion of positivity was superior to the number of arcs in the diagnosis of hydatidosis with the WHF antigen. However, the number of bands other than arc 5 may be of value in the diagnosis of hydatid disease by the IEP test in cases where this diagnostic arc is absent.

4194 THE EARLY DIAGNOSIS OF BILIARY ATRESIA. *(E.)* Kimura, S. (Iwate Med. Coll., Morioka, Japan). *Prog. Pediatr. Surg.* 6:91-112, 1974.

Thirty-four cases of biliary atresia and 12 cases of neonatal hepatitis were analyzed with regard to differential diagnosis. Seven cases of neonatal hepati-

tis and 22 patients with biliary atresia were admitted before the age of ten weeks. About 35% of the atresia cases while only about 25% of the hepatitis cases had jaundice present from birth. Marked progressive hepatomegaly was a characteristic of both groups, but a decrease in liver size before 3 months of age indicated hepatitis. Liver consistency was always harder in cases of atresia. Routine liver function tests were of little value. Serial determinations of total serum bilirubin levels showed increases in all but two of 10 cases of atresia, while hepatitis tended to show a fluctuating or gradually descending curve. High levels of alkaline phosphatase were found only in atresia during critical periods. Elevation of serum leucine aminopeptidase activity was more frequent in atresia than in hepatitis. The Rose Bengal test resulted in excretion of less than 6% ^{131}I in most of the atresia cases, and over 9% in most of the hepatitis cases during the 72 hr after i.v. administration. However, contamination of the feces with urine, despite precautions, reduced reliability. Serial scintigrams of the liver at 1, 24, 48 and 72 hr after injection of the ^{131}I proved to be more reliable. Shadows of the liver and kidneys could be clearly seen in atresia but not in hepatitis. Biopsy showed that portal fibrosis, ductular proliferation, and cholestasis in the ductular system and liver cells were more marked in biliary atresia. It is concluded that serial total bilirubin tests, leucine aminopeptidase activity tests, histologic examination of the liver, and most importantly the ^{131}I Rose Bengal excretion test with scintigraphy of the liver are most valuable in the differential diagnosis of these two conditions.

4195 EVALUATION OF BARIUM AND GASTROGRAFIN AS CONTRAST MEDIA FOR THE DIAGNOSIS OF ESOPHAGEAL RUPTURES OR PERFORATIONS. *(E.)* Vessal, K.; Montali, R. J.; Larson, S. M.; Chaffee, V.; James, A. E., Jr. (Pahlavi Univ. Med. Sch., Shiraz, Iran). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):307-319, 1975.

The effects of barium sulfate and meglumine diatrizoate, alone and in combination with bacterial cultures of pooled human mouth flora, on the feline mediastinum were evaluated. Catheters were passed through the abdominal esophagus into the mediastinum of adult domestic short-haired cats. Commercial barium, sterile barium sulfate, meglumine diatrizoate, and/or flora were injected into the mediastinum *via* the catheters and the animals were monitored clinically and with chest roentgenograms for 2-90 days. All cats injected with the commercial barium had well-localized white, chalky deposits within the paraesophageal mediastinal tissue at the sites of injection, and some animals had similar foci scattered throughout the visceral and intercostal pleura. Granulomatous inflammation including vascular tissue and fibrosis was observed, the extent of such lesions generally increasing with time; these lesions were free of bacterial, fungal, and protozoal organisms. Similar lesions were observed in the cats injected with sterile barium sulfate and in those injected with barium plus flora. Most of the cats injected with meglumine diatrizoate alone showed no pathologic changes, although 1 had a minimal inflammatory reaction; most of the cats injected with meglumine diatrizoate plus flor had mild inflammatory reactions. Flora alone produced mildly to moderately acute mediastinal inflammation. In cultures of human flora, the commercial barium inhibited growth by 70%, while the sterile barium sulfate inhibited growth by 55%. The data suggest that the water-soluble contrast media cause no significant histologic reactions in the mediastinum. Although barium causes granuloma formation, its superior physical properties make it as the contrast agent of choice with difficult clinical problems relating to esophageal rupture or perforation.

4196 VALUE OF THE ENDOSCOPIC DYING METHOD WITH METHYLENBLUE. *(E.)* Deyhle, P.; Tschen, H. (Dep. Inn. Med., Univ. Zurich, Switzerland). *Endoscopy* 7(1):24-26, 1975.

The specificity of the endoscopic methylene blue dye method described by Suzuki *et al.* was examined using 75 patients in whom diagnostic evidence had already been obtained. All of the carcinomas in these patients were homogeneously stained a deep blue, all stained areas yielding carcinomatous tissue on biopsy. The extent of the carcinoma was also more accurately defined after staining as compared with the results obtained with normal endoscopy. None of 12 benign ulcers took up the dye at their margins, and an adenomatous gastric polyp also failed to stain. Seventeen of 50 stomachs which appeared normal on normal endoscopy showed a stippled staining pattern with methylene blue which was readily distinguishable from the staining patterns of the carcinomas. Most of these areas showed intestinal metaplasia, although mucous-secreting cells were also stained. In 1 instance, an intestinal metaplasia failed to stain. In some cases, fragments of mucus adhering to the stomach wall took up the stain; in these instances, washing with water removed the dye. The results suggest that this dye method should be used where endoscopy points to suspected early cancer or to a carcinomatous ulcer, but where biopsy samples provide no confirmation. The method is, however, too time-consuming to find acceptance for routine endoscopy.

4197 DIFFERENTIATION OF PANCREATITIS FROM COMMON BILE DUCT OBSTRUCTION WITH HYPERAMYLASEMIA. *(E.)* Lesser, P. B.; Warshaw, A. L. (Harvard Med. Sch., Boston, Mass.). *Gastroenterology* 68(4):636-641, 1975.

Patients with abdominal pain and hyperamylasemia (37) were evaluated to determine which elements of the history, physical examination, and laboratory tests are most useful in the differentiation of pancreatitis from common bile duct obstruction with hyperamylasemia. The patients studied had a surgical or definite radiological diagnosis of acute pancreatitis (13 patients), common duct stone without pancreatitis (9 patients), or common duct stone with acute pancreatitis (15 patients). The single facet of the history with discriminatory value was the location

of the pain: patients with common duct stone had right upper quadrant pain more frequently than patients with common duct stone and acute pancreatitis or acute pancreatitis alone. Laboratory tests alone, serum amylase, urinary amylase, serum lipase, serum transaminase, bilirubin, and serum calcium were not reliable in the discrimination of pancreatitis from bile duct obstruction. The renal clearance of amylase, expressed as a percentage of creatinine clearance, clearly separated patients with acute pancreatitis and those with common duct stone and acute pancreatitis from patients with common duct stone without pancreatitis with no overlap of values. The mean amylase:creatinine clearance ratio for acute pancreatitis (9.8) and for common duct stone with acute pancreatitis (8.9) was significantly higher than for common duct stone without pancreatitis (3.2) and controls (3.1). In this study, an amylase:creatinine clearance ratio of greater than 5.5% indicated clinically important pancreatitis, and, conversely, a lower value indicated the absence of significant pancreatitis. It is suggested that this index would be useful in guiding the selection of patients for safe, early common duct exploration.

4198 GALACTOSE RETENTION: AN ALTERNATIVE TO THE BROMSULPHTHALEIN TEST. *(E.)* Sinniah, D. (R. Victoria Hosp., Belfast, Northern Ireland). *Singapore Med. J.* 15(4):273-275, 1974.

A study of galactose retention was performed in 12 control subjects and 38 patients to determine if the galactose and BSP retention tests give equivalent information. The patient group included eight with obstructive jaundice, five with cancer metastases in the liver, eight with infectious hepatitis, and 17 with cirrhosis. The galactose test was carried out after an 8-12 hr fast. Galactose (350 mg/kg) was administered i.v. as a sterile 25% solution over a period not exceeding 3 min. When half the volume had been given, a stopwatch was started and 30 min later, 0.1 ml of blood was taken from a finger tip for galactose determination. In 15 of 17 cirrhotic patients, a 45 min BSP retention test was carried out immediately after the galactose test. The galactose retention was normal in 87.5% of patients with biliary obstruction and four single cases of mucocele of the gall bladder, the Budd-Chiari syndrome, duodenal ulceration, and polycythemia. The mean galactose retention in the group of patients with obstructive jaundice was 31.6 mg/100 ml; in the group with liver metastases, 84.0 mg/100 ml; in the hepatitis patients, 48.2 mg/100 ml; and in the cirrhotic patients, 88.8 mg/100 ml. There was good correlation between the results of the galactose and the BSP retention tests in the 15 cirrhotic patients. It is suggested that the galactose test has a place in the group of liver function tests because it can be used as an aid in differentiating between parenchymatous and obstructive jaundice and also as a substitute for the BSP test in the presence of jaundice, or where there is a history of allergy to BSP.

4199 PERCUTANEOUS ASPIRATION BIOPSY OF THE PANCREAS UNDER ULTRASONIC GUIDANCE. *(E.)* Smith, E. H.; Bartrum, R. J., Jr.; Chang, Y. C.; D'Orsi, C. J.; Lokich, J.; Abbruzzese, A.; Dantono, J. (Harvard Med. Sch., Boston, Mass.). *N. Engl. J. Med.* 292(16):825-828, 1975.

Percutaneous aspiration biopsy of the pancreas under ultrasonic guidance was evaluated in 7 patients with suspected pancreatic tumors. In each case, the pancreatic mass was localized with a standard ultrasonic transducer. The standard transducer was then replaced with a modified instrument equipped with a central canal through which a 19-gauge needle was passed and directed along the path of the sound beam. A 23-gauge needle attached to an aspiration syringe was then directed through the 19-gauge needle and passed into the mass. In 5 cases, the aspirates provided cytologically conclusive evidence of carcinoma, and in a sixth patient the aspirate was strongly suggestive of neoplasia. None of the patients experienced subjective discomfort either during or after the percutaneous puncture procedure, and there was no objective clinical evidence of noteworthy patient injury. Although fine-needle biopsy is not expected to prolong life in patients with pancreatic tumors, it does eliminate the need for more complicated, uncomfortable, and hazardous diagnostic procedures. In many cases, exploratory surgery may be obviated. This method therefore appears to offer considerable value in overall patient management.

4200 RADIOISOTOPE SCINTIGRAPHY IN DIAGNOSIS OF BARRETT'S ESOPHAGUS. *(E.)* Berquist, T. H.; Nolan, N. G.; Stephens, D. H.; Carlson, H. C. (Mayo Clin., Rochester, Minn.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):401-411, 1975.

A new approach to the diagnosis of Barrett's esophagus is described. Patients suspected of having Barrett's esophagus were fasted and given 5 mCi Tc^{99m} pertechnetate i.v. Scintigraphic imaging was performed with the patient standing after 30 and 60 min. After the scan, the patients were given barium p.o. and a roentgenogram was taken in the upright position. In the normal person, the isotope is intensely concentrated by the stomach, there is a lack of radionuclide uptake by the esophagus, and evidence of activity is seen in the cardiac pool. In Barrett's esophagus, the roentgenogram shows an esophageal hiatal hernia and a stricture of the mid-esophagus. Activity is taken up by the stomach and esophagus to a level corresponding to that of the stricture. In 5 men with Barrett's esophagus, the diagnosis was made on the basis of an abnormal uptake of the isotope by the lower esophagus. In 1 additional patient who had a rare variant of Barrett's esophagus, Barrett's ulcer, the radioisotope was concentrated in the areas of esophageal ulceration. In each instance, the diagnosis was confirmed by tissue biopsy. Three patients with esophageal conditions other than Barrett's esophagus or Barrett's ulcer were also studied; scintigrams showed no esophageal uptake of pertechnetate and biopsies confirmed the absence of gastric mucosa in the esophagus. Scanning of the esophagus with Tc^{99m} pertechnetate appears to be useful in

making or excluding a diagnosis of Barrett's esophagus and promises to be useful in studying the etiology of the disorder.

4201 ENDOSCOPIC EXAMINATIONS IN SOME GASTROINTESTINAL DISEASES IN CHILDREN. *(Rus.)* Mazurin, A. V.; Barchunov, B. N.; Zaprudnov, A. M.; Barchunova, N. N.; Miroshnichenko, V. A.; Prokopova, I. V.; Barisheva, L. N.; Morokova, R. N.; Shinkarenko, N. I. (N.I. Pirogov Med. Inst., Moscow, USSR). *Pediatriia* (3):6-12, 1975.

4202 RADIOLOGICAL EXAMINATION OF THE GASTROINTESTINAL TRACT WITH TC 100 (BARIUM SULFATE FERTIG-SUSPENSION). *(Ger.)* Rosarius, C. (Munic. Hosp., Cologne-Merheim, Germany). *Therapiewochen* 25(4):396-399, 1975.

4203 GASTROINTESTINAL ENDOSCOPY: TODAY AND TOMORROW. *(Ger.)* Ottenjann, R.; Bartelheimer, W.; Busse, R.; Lux, G. (Munic. Hosp., Munich-Neuperlach, Germany). *Munch. Med. Wochenschr.* 117 (20):851-854, 1975.

4204 THE DOUBLE-CONTRAST UPPER GASTROINTESTINAL EXAMINATION IN THE JAPANESE STYLE. AN EXPERIENCE WITH 2,000 EXAMINATIONS. *(E.)* Gelfand, D. W. (Wayne State Univ. Sch. Med., Detroit, Mich.). *Am. J. Gastroenterol.* 63(3):216-220, 1975.

4205 COMPUTER AIDED DATA PROCESSING FOR DIGESTIVE ENDOSCOPY WITH THE HELP OF A CODE-SYSTEM. *(E.)* Dobronte, Z.; Nafradi, J.; Varro, V.; Benedek, S. (Univ. Med. Sch., Szeged, Hungary). *Endoscopy* 7(1):1-5, 1975.

4206 EMERGENCY ENDOSCOPY IN DIGESTIVE HEMORRHAGE. *(Sp.)* Ferraz Ramos, J. (Fac. Med., Montivideo, Uruguay). *Cir. Urug.* 44(4):274-277, 1974.

4207 RADIOLOGY IN THE STUDY OF DIGESTIVE HEMORRHAGES. *(Sp.)* Boschi, J. (Fac. Med., Montevideo, Uruguay). *Cir. Urug.* 44(4):270-273, 1974.

4208 EVALUATION OF ESOPHAGOGASTRODUODENOSCOPY IN DETECTION OF ACUTE GASTROINTESTINAL BLEEDING. *(Rus.)* Rozanov, B. S.; Rokhlin, E. A.; Dmitriev, A. N. (S. P. Botkin Hosp., Moscow, USSR). *Khirurgiia (Mosk.)* 616(9):88-95, 1974.

4209 UPPER GASTROINTESTINAL FIBERENDOSCOPY: AN ANALYSIS OF 100 CONSECUTIVE PROCEDURES. *(E.)* Bono, J. A. (VA Hosp., Salem, Va.). *Am. Surg.* 41(5):315-320, 1975.

4210 DIAGNOSIS OF AN ENTERIC DUPLICATION WITH PERTECHNETATE ^{99m}Tc SCANNING. *(E.)* Schwesinger, W. H.; Croom, III, R. D.; Habibian, M. R. (Univ. Texas Health Sci. Cent., San Antonio). *Ann. Surg.* 181(4):428-430, 1975.

4211 HEMODYNAMIC PARAMETERS AND THE ELECTROCARDIOGRAM IN ESOPHAGOGASTRODUODENOSCOPY. *(Rus.)* Dubova, M. N.; Belov, I. N.; Mazhenov, A. S.; Struchkov, Iu. V. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* 616(4):89-95, 1974.

4212 GENERAL RADIOLOGIC AND ANGIOGRAPHIC DIAGNOSIS IN BLEEDING OF SMALL AND LARGE INTESTINES AND RECTUM. *(Ger.)* Wenz, W. (Inst. Radiol. Diagn., Univ. Freiburg, Germany). *Langenbecks Arch. Chir.* 337(Suppl.):563-568, 1974.

4213 ECHOGRAPHY IN THE DIAGNOSIS OF ABNORMALITIES OF THE LIVER, GALLBLADDER AND PANCREAS. *(E.)* Koch, C. W. (no affil.). *Radiol. Clin. Biol.* 44(2):149, 1975.

4214 SERUM ARYLAMIDASE ISOENZYMES IN DISEASES OF THE PANCREAS AND LIVER. *(Ger.)* Klein, V. E.; Mackensen, S. (Med. Clin. Univ. Kiel, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:544-546, 1974.

4215 THE ACIDOTEST TECHNIQUE FOR CHILDREN. *(Rus.)* Solntseva, A. A.; Nozdrina, R. A.; Totchasova, G. V.; Smirnova, S. V.; Maksimova, V. G. (Ivanovo Med. Inst., USSR). *Lab. Delo* (3):137-138, 1975.

4216 DYSPHAGIA AND ESOPHAGOSCOPY: THE USE OF THE FLEXIBLE FIBER ESOPHAGOSCOPE FOR THE DIAGNOSIS OF TUMORS IN THE DISTAL PORTION OF THE ESOPHAGUS AND IN THE CARDIA. *(Fl.)* Flendrig, J. A.; Brinkman, W. F. B.; Driessen, W. M. M.; van Tongeren, J. H. M. (St. Radboud Hosp., Nijmegen, Netherlands). *Ned. Tijdschr. Geneeskd.* 118(38): 1433-1438, 1974.

4217 INTRAESOPHAGEAL PRESSURE MEASUREMENTS IN THE DIFFERENTIAL DIAGNOSIS OF DYSPHAGIA. *(Ger.)* Wienbeck, M. (Med. Clin., Univ. Dusseldorf, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:353-360, 1974.

4218 BENIGN INTRALUMINAL STENOSIS OF THE ESOPHAGUS. *(Ger.)* Koischwitz, D.; Sobbe, A.; Miederer, S. E.; Stadelmann, O. (Clin. Radiol., Univ. Bonn, Germany). *Fortschr. Geb. Roentg. str. Nuklearmed.* 121(1):161-175, 1974.

4219 USE AND ABUSE OF GASTRIC FUNCTION TESTS BY BRITISH AND AMERICAN GASTRIC SURGEONS. *(E.)* Baron, J. H.; Griffen, Jr., W. O.; Alexander-Williams, J. (R. Postgrad. Med. Sch., London, England). *Am. J. Dig. Dis.* 20(4):370-376, 1975.

4220 THE SURFACE PATTERN OF THE STOMACH. *(E.)* Kreel, L. (Northwick Park Hosp., Clin. Res. Cent., Harrow, England). *Proc. R. Soc. Med.* 68(2):111-114, 1975.

4221 DIMETHYLPOLYSILOXAN AS AN AUXILLARY AID IN ENDOSCOPY. *(Ger.)* Gobel, D. (Richt. Hosp., Karlsruhe, Germany). *Leber Magen Darm* 4(4):174-176, 1974.

4222 GASTROSCOPIC DIAGNOSIS OF GASTRIC POLYPS. *(Ger.)* van Husen, N.; Manitz, G. (Med. Clin., Univ. Munster, Germany). *Med. Welt* 25(31-32):1280-1282, 1974.

4223 STUDIES ON INDIRECT MASS RADIOGRAPHIC EXAMINATIONS OF THE STOMACH. *(Jpn.)* Miura, T.; Hachiya, T.; Takahashi, H.; Ohishi, H.; Hamada, N.; Shibatsuji, H.; Aburatani, R.; Shungawa, M.; Kondo, H. (Nara Med. Univ., Japan). *J. Nara Med. Assoc.* 25(3):201-212, 1974.

4224 POSSIBILITY OF COMBINING FIBERGASTROSCOPIC AND CYTOLOGIC INVESTIGATIONS IN THE DIAGNOSIS OF GASTRIC CANCER. *(Rus.)* Pavlov, K. A.; Stefanenko, J. F.; Iudkovskaia, I. L.; Puchkov, J. G. (N. N. Petrov Res. Inst. Oncol., Leningrad, USSR). *Vopr. Onkol.* 21(1):39-45, 1975.

4225 RADIOLOGICAL EXAMINATION OF THE STOMACH WITH A POTENTIATED DOUBLE CONTRAST TECHNIQUE. *(E.)* Catalano, D. (St. Gennaro Hosp., Naples, Italy). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 121 (2):175-178, 1974.

4226 A STANDARDIZED METHOD FOR PHYSIOLOGICAL STIMULATION OF GASTRIN RELEASE. *(Ger.)* von Berger, L.; Raptis, S.; Dollinger, H.; Pfeiffer, E. F. (Cent. Intern. Med. Pediatr., Univ. Ulm, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:513-515, 1974.

4227 ENDOSCOPIC CRITERIA OF THE CURE OF ULCERS. *(It.)* Celli, L.; Santagostino, C. (Maggiore Hosp., Milan, Italy). *Clin. Eur.* 13(2):84-93, 1974.

4228 RADIOLOGICAL DIAGNOSIS OF POSTBULBAR DUODENAL ULCER BY HYPOTONIC DUODENOGRAPHY. *(Jpn.)* Miura, T.; Ohishi, H.; Takahashi, H.; Hachiya, T.; Sunagawa, M.; Aburatani, R.; Hamada, N.; Shibatsuji, H. (Nara Med. Univ., Japan). *J. Nara Med. Assoc.* 25(3):241-248, 1974.

4229 FUNCTIONAL ENDOSCOPY IN THE DUODENUM. *(E.)* Nakajima, M.; Murakami, K.; Kohli, Y.; Kawai, K. (Kyoto Prefect. Univ. Med., Japan). *Am. J. Gastroenterol.* 63(3):240-245, 1975.

4230 ENDOSCOPIC DIAGNOSIS OF DUODENAL DISEASES. *(Ger.)* Iwamura, K.; Saito, T. (Med. Clin., Univ. Tokai, Tokyo, Japan). *Leber Magen Darm* 4(5): 235-241, 1974.

4231 THE USE OF D-XYLOSE FOR THE STUDY OF INTESTINAL ABSORPTION FUNCTION IN NORMAL YOUNG CHILDREN AND IN CHRONIC NUTRITIONAL DISORDERS. *(Rus.)* Titova, L. S.; Patarakina, A. A.; Beier, L. V.; Sokoloba, A. S.; Serebriakova, N. N.; Golovina, N. E. (Gorkii Inst. Pediatr., USSR). *Pediatriia* (10):27-29, 1974.

4232 THE DIAGNOSTIC VALUE OF ALKALINE PHOSPHATASE FRACTIONS IN HUMAN SERUM. *(Ger.)* van Husen, N.; Gerlach, U. (Med. Clin., Univ. Munster, Germany). *Dtsch. Ges. Inn. Med.* 80:496-499, 1974.

4233 RADIOLOGICAL DIAGNOSIS OF DISEASES OF THE DUODENUM AND PANCREAS BY SIMPLIFIED HYPOTONIC DUODENOGRAPHY. *(Jpn.)* Ohishi, H.; Hatiya, T.; Sunagawa, M.; Shibatsuji, H.; Aburatani, R.; Takahashi, H.; Hamada, N.; Miura, T.; Inoue, K.; Ohtani, Y.; Matuyoshi, M.; Matuyoshi, T.; Nishigori, S. (Nara Med. Univ., Japan). *J. Nara Med. Assoc.* 25(3):249-257, 1974.

4234 CONTRIBUTION TO THE TECHNIQUE OF HYPOTONIC DUODENOGRAPHY. *(Ser.)* Ojdanic, Z.; Nadj, G. (Munic. Clin. Hosp., Belgrad, Yugoslavia). *Srp. Arh. Celok. Lek.* 102(3/4):217-228, 1974.

4235 ARTERIOVENOUS MALFORMATIONS OF THE BOWEL. AN OBSCURE CAUSE OF BLEEDING. *(E.)* Crichlow, R. W.; Mosenthal, W. T.; Spiegel, P. K.; House, R. K. (Dartmouth-Hitchcock Med. Cent., Hanover, N.H.). *Am. J. Surg.* 129(4):440-448, 1975.

4236 FIBEROPTIC COLONOSCOPY AND CYTOLOGY. *(E.)* Kline, T. S.; Yum, K. Y. (Lankenau Hosp., Philadelphia, Pa.). *Lab. Invest.* 32(3):428,1975.

4237 COLONOSCOPY. *(E.)* Overholt, B. F. (St. Mary's Mem. Hosp., Knoxville, Tenn.). *Cancer* 25(2):74-81, 1975.

4238 DIAGNOSTIC POSSIBILITIES WITH FIBEROPTIC COLONOSCOPY. *(Rus.)* Federov, V. D.; Pokrovskii, G. A.; Strekalovskii, V. P. (Res. Inst. Proctol. Lab. Clin., Moscow, USSR). *Khirurgiia (Mosk.)* 616(9):101-106, 1974.

4239 BIOPSY IN COLONOSCOPY. *(Fr.)* Chabanon, R. (Eduoard Herriot Hosp., Lyon, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(4):279-284, 1974.

4240 PREPARATORY PROCEDURE FOR DOUBLE CONTRAST X-RAY EXAMINATION OF THE COLON. *(Ger.)* Hauger, W. (Inst. Med. Radiol., Univ. Tubingen, Germany). *Leber Magen Darm* 4(3):125-130, 1974.

4241 CYTOLOGICAL TECHNIQUE FOR THE DIAGNOSIS OF SOME DISEASES OF THE DISTAL LARGE INTESTINE. *(Rus.)* Kravchuk, I. P.; Baranova, K. F. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Klin. Med. (Mosk.)* 53(3):78-83, 1975.

4242 SPONGIOPSY [RECTAL BIOPSY PLACED ON A GEL FOAM SPONGE]. *(Fr.)* Richir, C.; Soullard, J. (Bichat Hosp., Paris, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(4):285-289, 1974.

4243 COLON CLEANSING BY WYLAXINE AS PREPARATION FOR BARIUM ENEMA FLUOROSCOPY. *(Fr.)* Brombart, M.; Massun, P. (Inst. Radiol. Diagn., Brussels, Belgium). *Acta Gastroenterol. Belg.* 37(7-8):364-372, 1974.

4244 CARCINOEMBRYONIC ANTIGEN (CEA) LEVELS AND TUMOR HISTOLOGY IN COLON CANCER. *(E.)* Bivins, B. A.; Meeker, Jr., W. R.; Griffen, Jr., W. O. (Univ. Kentucky Med. Cent., Lexington). *J. Surg. Res.* 18(3):257-261, 1975.

4245 METHODS FOR INVESTIGATING THE PANCREAS AND THEIR VALUE IN THE CHILDREN'S HOSPITAL. *(Rus.)* Livshits, E. G. (Riga Med. Inst., USSR). *Pediatriia* (3):67-71, 1975.

4246 VALUE OF THE PROVOCATIVE TEST AND FECAL CHYMOTRYPSIN AND FAT DETERMINATIONS IN THE DIAGNOSIS OF PANCREATIC DISEASE. *(Ger.)* Sommer, H.; Kasper, H.; Chimombe, H. E. (Med. Clin., Univ. Wurzburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:548-549, 1974.

4247 CONTROLLED STUDIES ON THE DIAGNOSTIC VALUE OF FECAL CHYMOTRYPSIN DETERMINATIONS. *(Ger.)* Durr, H. K.; Otte, M.; Forell, M. M.; Bode, J. C. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:546-548, 1974.

4248 CATHETERIZATION OF THE PAPILLA THROUGH THE DUODENUM: POSSIBILITIES AND LIMITATIONS. *(Ger.)* Classen, M.; Koch, H.; Demling, L. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:350-353, 1974.

4249 PANCREAS SCINTIGRAPHY WITH A SCINTILLATION CAMERA. *(Ger.)* Markwardt, J.; Nauber, G.; Oczko, B. (Robert Rossle Clin., Berlin, Germany). *Radiol. Diagn. (Berl.)* 15(1):83-91, 1974.

4250 DOES THE COMBINATION OF PANCREATIC SCINTIGRAPHY AND HYPOTONIC DUODENOGRAPHY IMPROVE DIAGNOSTIC INFORMATION? *(Ger.)* Luschnitz, E.; Taubert, W.; Lange, H. (Dep. Med., Karl Marx Univ., Leipzig, Germany). *Radiol. Diagn. (Berl.)* 15 (1):93-97, 1974.

4251 CLINICAL EVALUATION OF SURGICAL PANCREATOGRAPHY. *(Rus.)* Shamanov, A. Kh. (Clin. Hosp., No. 40, Kharkov, USSR). *Klin. Khir.* (4):46-48, 1975.

4252 DIAGNOSIS OF PANCREATIC DISEASE BY ENDOSCOPIC PANCREATOGRAPHY. *(Jpn.)* Sunagawa, M.; Ohishi, H.; Hachiya, T.; Takahashi, H.; Aburatani, R.; Shibatuzi, H.; Kondo, H.; Hamada, N.; Takahashi, A.; Fukuyama, T.; Miura, T. (Nara Med. Univ., Japan). *J. Nara Med. Assoc.* 25(4/5):350-354, 1974.

4253 SERUM LIPASE ACTIVITY AND ENDOSCOPIC RETROGRADE PANCREATOGRAPHY IN CHRONIC PANCREATITIS AND PANCREATIC NEOPLASMS. COMPARITIVE STUDIES. *(Ger.)* Bornschein, W.; Weib, H. D. (2nd Med. Clin., Tech. Coll., Munich, Germany). *Dtsch. Med. Wochenschr.* 100(7):297-308, 1975.

4254 INDICATIONS AND TECHNICS IN SURGICAL THERAPY OF CHRONIC PANCREATITIS. *(Ger.)* Schwemmle, K. (Surg. Clin. Polyclin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Chirurg* 45(10):465-470, 1974.

4255 INDICATIONS, TECHNICAL APPROACH, DIAGNOSTIC VALUE AND RISKS OF ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATICOGRAPHY (ERCP). *(Ger.)* Miederer, S. E.; Stadelmann, O.; Loffler, A.; Wobser, E.; Koischwitz, D. (Med. Polyclin., Univ. Bonn, Germany). *Leber Magen Darm* 4(5):187-194, 1974.

4256 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATICOGRAPHY. *(Cz.)* Skala, I.; Pirk, F.; Skala, J. (Res. Cent. Metab. Nutr., Prague, Czechoslovakia). *Rozhl. Chir.* 53(7):437-446, 1974.

4257 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY AS A POSTOPERATIVE INVESTIGATIVE TECHNIQUE. *(Ger.)* Miederer, S. E.; Loffler, A.; Siedek, M.; Stadelmann, O. (Med. Polyclin., Univ. Bonn, Germany). *Med. Welt* 25(31-32):1285-1287, 1974.

4258 HAZARDS OF ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATICOGRAPHY (ERCP). *(E.)* Classen, M.; Demling, L. (Dep. Med., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):1-3, 1975.

4259 PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY (PTC). *(E.)* Schoolmeesters, J. E. P. V. (no affil.). *Radiol. Clin. Biol.* 44(2):147-148, 1975.

4260 INDOCYANINE GREEN CLEARANCE - A LIVER FUNCTION TEST. *(E.)* Brugmann, E.; Towe, J. (Med. Clin., Univ. Rostock, Germany). *Mater. Med. Pol.* 6(2):123-126, 1974.

4261 RETROSTERNAL LYMPHOGRAPHY. *(Ger.)* Meyer-Burg, J.; Schworer, I.; Arbeiter, G. (Med. Clin. Polyclin., Free Univ. Berlin, Germany). *Leber Magen Darm* 4(6):291-298, 1974.

4262 DIAGNOSTIC VALUE OF SERUM VITAMIN A (RETINOL) AND RETINOL-BINDING PROTEINS. *(Ger.)* Olbermann, M.; Prellwitz, W.; Kapp, S.; Muller, D. (Med. Clin., Univ. Mainz, Germany). *Dtsch. Ges. Inn. Med.* 80:491-494, 1974.

4263 IMPORTANCE OF THE DETERMINATION OF ALKALINE PHOSPHATASE ISOENZYMES IN DIAGNOSIS. *(Cz.)* Komarkova, A.; Bilyk, I. (Fac. Hosp., Kunz, Prague, Czechoslovakia). *Chem. Listy* 69(1):59-69, 1975.

4264 FIBEROPTIC ENDOSCOPY IN THE DIAGNOSIS OF UPPER GASTROINTESTINAL BLEEDING ASSOCIATED WITH CIRRHOSIS. *(Sp.)* Herrerias, J. M.; Lopez Sanchez, A.; Sanchez Venegas, S. (Fac. Med., Univ. Seville, Cadiz, Spain). *Rev. Esp. Enferm Apar. Dig.* 45(3):307-316, 1975.

4265 ENZYME DETERMINATIONS IN BIOPSY CYLINDERS FOR DIFFERENTIAL DIAGNOSIS OF CHRONIC HEPATITIS. *(Ger.)* Maier, K. P.; Volk, B.; Gero , W. (Med. Clin., Univ. Freiburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:499-501, 1974.

4266 THE VALUE OF STAINING RETICULIN FIBERS IN THE DIAGNOSIS OF LIVER DISEASE. *(Ser.)* Budakov, P.; Kollin, J. (Clin. Hosp., Novi Sad, Yugoslavia). *Med. Pregl.* 27(11/12):447-450, 1974.

4267 THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE IN SURGICAL HOSPITALS. *(Rus.)* Furmanenko, E. D.; Ishchuk, O. E.; Chubar, S. V.; Shalimov, S. A. (Kiev Res. Inst. Clin. Exp. Surg., USSR). *Klin. Khir.* (4):38-41, 1975.

4268 ENZYME CHANGES IN TWO GROUPS OF JAUNDICED PATIENTS (EXTRAHEPATIC OBSTRUCTION AND VIRAL HEPATITIS) COMPARED. *(E.)* Chua, K. L.; Seah, C. S. (Thomson Road Gen. Hosp., Singapore). *Singapore Med. J.* 15(4):246-252, 1974.

4269 DIAGNOSTIC VALUE OF LAPAROSCOPY IN HEPATOMA: 60 CASES. *(Fr.)* Debray, C.; Paolaggi, J. A.; Mugnier, B.; Barret, C. (Bichat Hosp., Paris, France). *Ann. Med. Interne* 126(4):273-277, 1975.

4270 SELECTIVE ANGIOGRAPHY OF THE ABDOMINAL AORTA BRANCHES IN THE DIAGNOSIS OF SPACE-OCCUPYING DISEASES OF THE LIVER. *(Rus.)* Korepanov, V. I.; Zhuravlev, V. A.; Kolupaeva, G. N. (Kirov Res. Inst. Blood Transfus., USSR). *Khirurgiia (Mosk.)* 616(3):69-73, 1974.

4271 RADIOLOGY OF THE LIVER. *(Ger.)* Wenz, W. (Inst. Radiol. Diagn., Univ. Freiburg, Germany). *Tijdschr. Gastroenterol.* 17(1):24-39, 1974.

4272 THE VALUE OF BILIARY ENDOSCOPY. *(E.)* Shore, J. M.; Berci, G.; Morgenstern, L. (Cedars-Sinai Med. Cent., Los Angeles, Calif.). *Surg. Gynecol. Obstet.* 140(4):601-604, 1975.

4273 DIAGNOSTICS OF THE BILIARY TRACT. *(E.)* Frommhold, W. (Leiden Univ. Hosp., Netherlands). *Radiol. Clin. Biol.* 44(2):146-150, 1975.

4274 PATHOLOGICAL ENDOSCOPIC FINDINGS AT THE PAPILLA OF VATER AND AT THE PREPAPILLAR CHOLEDOCHUS. *(Ger.)* Seifert, E.; St. Stender, H.; Lesch, P.; Luska, G. (Med. Coll. Hannover, Germany). *Leber Magen Darm* 4(5):226-234, 1974.

4275 ADENOMYOMATOSIS AS A VARIETY OF HYPERPLASTIC CHOLECYSTOSIS. *(E.)* Wissink, H.; Rosenbusch, G. (no affil.). *Radiol. Clin. Biol.* 44(2):147, 1974.

4276 EXPERIENCE WITH 1179 CHOLECYSTANGIOGRAPHIES. *(Ger.)* Holsten, D. R.; Husmann, K. H. (Silvoah City Hosp., Hannover, Germany). *Roentgenpraxis* 27(9):469-471, 1974.

4277 SPECIAL RADIOLOGIC DIAGNOSIS OF THE EXTRAHEPATIC BILE DUCTS. *(Ger.)* Frommhold, H. (Clin. Radiol., Univ. Bonn, Germany). *Roentgenpraxis* 27(5):218-225, 1974.

4278 RATIONAL DIAGNOSIS OF THE GALLBLADDER AND BILE DUCTS. *(Ger.)* Taenzer, V. (Clin. Radiol. Nucl. Med. Phys. Ther., Free Univ. Berlin, Germany). *Roentgenpraxis* 27(5):213-217, 1974.

4279 COMPARITIVE CLINICAL TRIAL OF A NEW INTRAVENOUS CONTRAST MEDIUM, B 10610, AND OF THE SODIUM AND MEGLUCAMINE SALTS OF IOGLYCAMIC ACID IN RADIOLOGICAL EXAMINATION OF THE BILIARY TRACT. *(It.)* Corsi, M.; Tassini, T. (Munic. Hosp., Udine, Italy). *Quad. Radiol.* 38(4):261-274, 1973.

4280 PROJECTION VARIANTS IN RADIOLOGICAL EXAMINATION OF THE GALLBLADDER REFLEX. *(Ger.)* Reifmann, V. K.; Holzel, W. (Radiol. Clin., Martin Luther Univ., Halle-Wittenberg, Germany). *Z. Gesamte. Inn. Med.* 30(3):115-117, 1975.

4281 COMMENTS ON THE EVALUATION OF PEROPERATIVE CHOLANGIOGRANS. *(Cz.)* Krysl, I. (Fac. Med. Hyg., Charles Univ., Prague, Czechoslovakia). *Rozhl. Chir.* 53(7):447-449, 1974.

4282 ADVANCES IN THE IMMUNODIAGNOSIS OF PARASITIC INFECTIONS. *(E.)* Kagan, I. G. (Cent. Dis. Control, Atlanta, Ga.). *Z. Parasitenk.* 45(2):163-195, 1974.

4283 USE OF RADIOIMMUNOASSAY IN ENDOCRINE GI TUMOURS. *(E.)* Walsh, J. H. (no affil.) *Clin. Gastroenterol.* 3(3):643-655, 1974.

4284 THE ROLE OF RADIOLOGY IN THE DETECTION OF ENDOCRINE TUMOURS IN THE GI TRACT. *(E.)* Cope, V.; Warwick, F. (no affil.). *Clin. Gastroenterol.* 3(3):621-642, 1975.

4285 HISTOLOGICAL DIAGNOSIS OF VIRAL HEPATITIS. *(E.)* Desmet, V. J.; De Groote, J. (no affil.). *Clin. Gastroenterol.* 3(2):337-354, 1974.

4286 RADIOLOGICAL CONCEPTS OF CHOLECYSTOKININ CHOLECYSTOGRAPHY. *(E.)* Parks, M. P. (no affil.). *Radiol. Technol.* 46(5):335-342, 1975.

4287 ENDOSCOPIC CHOLANGIOGRAM AND INTRAVENOUS CHOLANGIOGRAM: AN ANALYSIS OF SIZE COMPARISON. *(E.)* Belsito, A. A.; Marta, J. B.; Dickinson, P. B.; Pizinger, R. J. (United Hosp., St. Paul, Minn.). *Gastrointest. Endosc.* 21(4):183, 1975.

4288 ENDOSCOPIC RETROGRADE CHOLANGIOGRAM (ERC) IN THE EVALUATION OF NON-JAUNDICED PATIENTS WITH BILIARY TYPE SYMPTOMS. *(E.)* Belsito, A. A.; Marta, J. B.; Dickinson, P. B.; Pizinger, R. J. (United Hosp., St. Paul, Minn.). *Gastrointest. Endosc.* 21(4):183, 1975.

4289 ENDOSCOPIC SAMPLING FOR TISSUE DIAGNOSIS IN GASTROINTESTINAL MALIGNANCY. *(E.)* Bemvenuti, G. A.; Hattori, K.; Levin, B.; Kirsner, J. B.; Reilly, R. W. (Dep. Med., Univ. Chicago, Ill.). *Gastrointest. Endosc.* 21(4):159-161, 1975.

4290 GASTRIC MYELOMA. *(E.)* Balthazar, E. J.; Cano, R. (New York Med. Coll. Flower-Fifth Ave. Hosp., N.Y.). *Am. J. Gastroenterol.* 63(4):340-344, 1975.

4291 PEDIATRIC ENDOSCOPY WITH FLEXIBLE FIBERSCOPES. *(E.)* Gans, S. L.; Ament, M.; Christie, D. L.; Liebman, W. M. (Cedars Sinai Med. Cent., Univ. California, Los Angeles). *J. Pediatr. Surg.* 10(3):375-380, 1975.

4292 LIVER FUNCTION. *(E.)* Burke, M. D. (Univ. Minnesota Sch. Med., Minneapolis). *Hum. Pathol.* 6(3):273-286, 1975.

4293 XANTHOMA OF THE GASTRIC AND DUODENAL MUCOSA. *(E.)* Brodmerkel, Jr., G. J.; Agrawal, R. M.; Cincala, R. P.; Chandrasekaran, S. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Gastrointest. Endosc.* 21(4):184, 1975.

4294 THE FEATURES OF THE INTRAHEPATIC CHOLANGIOGRAM IN RELATION TO LIVER PATHOLOGY ESTABLISHED ON BIOPSY. *(E.)* Ayoola, E.; Vennes, J. A.; Rohrmann, C. A.; Silvis, S. E. (Minneapolis VA Hosp., Minn.). *Gastrointest. Endosc.* 21(4):183, 1975.

4295 CLINICAL EVALUATION OF SPHINCTER OF ODDI MANOMETRY. *(E.)* Bradley, V. D.; Messian, R. A.; Fornes, M. F.; Nebel, O. T. (Nav. Reg. Med. Cent., San Diego, Calif.). *Gastrointest. Endosc.* 21(4):184, 1975.

4296 GB SIGN: AN ENDOSCOPIC CLUE TO CHRONIC CHOLECYSTITIS. *(E.)* Brodmerkel, Jr., G. J.; Agrawal, R. M.; Cincala, R. P.; Chandrasekaran, S. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Gastrointest. Endosc.* 21(4):184, 1975.

4297 COMPUTER-AIDER DIAGNOSIS OF "DYSPEPSIA". *(E.)* Horrocks, J. C.; de Dombal, F. T. (Gen. Infirm., Leeds, England). *Am. J. Dig. Dis.* 20(5):397-406, 1975.

4298 CLINICAL CYTOLOGY OF THE LIVER. *(E.)* Carney, C. N. (Univ. North Carolina, Sch. Med., Chapel Hill). *Acta Cytol. (Baltimore)* 19(3): 244-250, 1975.

See also, 4316, 4317, 4340, 4373, 4586, 4590, 4645, 4651, 4666, 4708, 4724, 4787, 4808, 4809, 4810, 4817, 4821, 4822, 4823, 4824, 4869, 4911, 4943, 5022, 5115, 5120.

GASTROINTESTINAL DISEASES

ESOPHAGUS

4299 EFFECT OF INCREASED INTRAABDOMINAL PRESSURE ON LOWER ESOPHAGEAL SPHINCTER PRESSURE. *(E.)* Dodds, W. J.; Hogan, W. J.; Miller, W. N.; Stef, J. J.; Arndorfer, R. C.; Lydon, S. B. (Med. Coll. Wisconsin, Milwaukee). *Am. J. Dig. Dis.* 20(4):298-308. 1975.

Lower esophageal sphincter (LES) responses to increased intra-abdominal pressure were evaluated in 20 normal subjects (mean age 22.6 yr) and 35 patients (mean age 47.3 yr) with reflux esophagitis. Intra-abdominal and intragastric pressure were increased by abdominal compression, the Valsalva maneuver, and leg raising. Changes in LES pressure were measured by station and rapid pull-through techniques, using a catheter system capable of radially recording pressure. Abdominal compression and Valsalva increased LES pressure in normal subjects, but the increase was about equal to the gastric pressure increase. Therefore, the LES-gastric pressure gradient remained unchanged. During leg raising, the LES pressure increased more than gastric pressure, thereby increasing the LES-gastric pressure gradient. Although statistically significant, the LES pressure increase (to 32.7 mm Hg) induced by leg raising was modest, unrelated to initial sphincter pressure, and unaffected by atropine. Excluding six individuals demonstrating a "common cavity" phenomenon, LES pressure changes during abdominal compression were similar in patients with esophagitis and controls. Consequently, LES response to abdominal compression does not separate most patients with esophagitis from normal subjects. The observed LES responses to increased intra-abdominal pressure are analyzed in terms of two mechanisms: extrinsic mechanical factors and a physiologic adaptive response of intrinsic LES tone. It is concluded that the findings are best accounted for by extrinsic mechanical factors.

4300 OROPHARYNGEAL DYSPHAGIA: MANOMETRIC AND CINE ESOPHAGRAPHIC FINDINGS. *(E.)* Hurwitz, A. L.; Nelson, J. A.; Haddad, J. K. (Fort Miley VA Hosp., San Francisco, Calif.). *Am. J. Dig. Dis.* 20(4):313-324, 1975.

The manometric and radiographic findings in a group of patients with oropharyngeal dysphagia were systematically studied and correlated. Manometric studies and cine-esophagrams were performed on 14 patients with symptoms of orophayrngeal dysphagia and 15 controls with a variety of other esophageal symptoms, usually related to acid reflux. In the controls, the mean peak pharyngeal pressure was 36 mm Hg; the mean upper-esophageal sphincter (UES) resting pressure was 39 mm Hg; UES relaxation to baseline esophageal pressure was complete in 95% of the swallows; and coordinated UES activity was observed in 98% of the swallows. The oropharyngeal dysphagia patients did not differ significantly from the controls with regard to peak pharyngeal pressures, but the mean UES resting pressure, percent UES relaxation, and percent UES coordination were significantly lower in the former. The cine-esophagrams showed abnormal mouth and tongue movements in seven patients, anatomically disorganized pharyngeal contraction in seven, abnormal UES relaxation in eight, and aspiration in five; initiation of a primary esophageal wave was normal in all but one patient. Peak pharyngeal monometric pressures did not correlate with the cine appearance of pharyngeal contraction; visual observations of the UES did not correlate well with manometric pressures; and the amount and site of aspiration did not correlate with the manometric measurements. However, manometry and cine study agreed in the evaluation of UES relaxation, UES coordination, and evaluation of primary peristalsis. Five patients underwent UES myotomy for severe intractable dysphagia, aspiration, and weight loss; although four patients improved clinically after surgery, the mean and maximum resting UES pressures and peak pharyngeal pressure did not change significantly. The data suggest that both cine-esophagography and manometry are necessary to properly evaluate the abnormalities associated with oropharyngeal dysphagia.

4301 EFFECT OF BILE SALTS ON THE IONIC PERMEABILITY OF THE ESOPHAGEAL MUCOSA AND THEIR ROLE IN THE PRODUCTION OF ESOPHAGITIS. *(E.)* Safie-Shirazi, S.; DenBesten, L.; Zike, W. L. (Univ. Iowa Coll. Med., Iowa City). *Gastroenterology* 68(4):728-733, 1975.

The effects of HCl and HCl plus bile salts on ionic movement across the mucosa of the distally occluded canine and human esophagus were measured to test the hypothesis that HCl, in concentrations secreted by the gastric mucosa, does not significantly penetrate or injure the esophageal mucosa unless its permeability to H+ is altered. Double lumen oroesophageal tubes were placed in the esophaguses of anesthetized mongrel dogs, after which the gastroesophageal junction was occluded and the esophagus was ligated just distal to the thyroid cartilage. A test solution (100 ml) containing 50 mEq/liter HCl with or without 2.5 mM bile salts was instilled into the proximally and distally occluded esophagus, where it was left for 0.5 or 4.5 hr. In two conscious human volunteers, the distal esophagus was occluded and 30 ml of test solution containing 100 mEq/liter HCl with or without 10 mM bile salts was instilled into the esophagus and left for 15 min. In the dogs there was a significant loss of H+ when bile salts were added to the test solution containing HCl. With HCl plus bile salts, but not HCl alone, a severe necrotizing esophagitis with loss of mucosa and submucosa was observed when the solution was left in the esophagus for 4.5 hr. In the human volunteers, the mean net losses of H+ from the esophagus increased five-fold in the presence of bile salts. The data indicate that the addition of bile to HCl significantly increases the loss of H+ and renders the esophageal mucosa more susceptible to inflammatory change. These observations suggest that the esophagitis and ulceration associated with intra-esophageal bile and HCl may be due to increased permeability of the esophageal mucosa to H+ induced by bile salts.

4302 CLINICAL TRIAL OF AN ANTACID IN DISEASES OF THE ESOPHAGUS, STOMACH, AND DUODENUM. *(Fr.)* ben Bouali, A.; Joubaud, F. (Clin. C. H. U. Angers, France). *Ouest Med.* 27(18):1703-1708, 1974.

4303 SURGICAL TREATMENT OF ESOPHAGEAL DISEASES. *(Rus.)* Shalimov, A. A.; Avilova, O. M.; Shalimov, S. A. (Kiev Res. Inst. Clin Exp. Surg., USSR). *Klin. Khir.* (4):25-31, 1975.

4304 PRELIMINARY TRIALS WITH ESOPHAGOJEJUNOPLICATION. *(Ger.)* Siewert, R.; Peiper, H. J.; Wallat, H. (Clin. Gen. Surg., Univ. Gottingen, Germany). *Bruns Beitr. Klin. Chir.* 221(5):343-354, 1974.

4305 FUNCTIONAL DISORDERS OF THE ESOPHAGUS AND GASTRIC CARDIA. *(Ger.)* Berndt, H. (Cent. Inst. Cancer Res., Robert Rossle Clin., Berlin-Busch, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4): 169-176, 1974.

4306 CLINICAL RESULTS OF FUNDOPLICATION. *(Ger.)* Siewert, R.; Wallat, H. J.; Krtsch, H.; Peiper, H. J. (Clin., Univ. Gottingen, Germany). *Langenbecks Arch. Chir.* 338(1):9-26, 1975.

4307 THE MANAGEMENT OF SYMPTOMATIC SCHATZKI RING. A REPORT OF 7 CASES. *(E.)* Hill, M.; Knauer, C. M. (Santa Clara Valley Med. Cent., San Jose, Calif.). *Gastrointest. Endosc.* 21(3): 116-118, 1975.

4308 ABDOMINAL APPRAOCH FOR THE LIGATION OF BLEEDING OESOPHAGEAL VARICES. *(E.)* Miller, S. S. (Dep. Surg., Univ. Aberdeen, Scotland). *Br. J. Surg.* 62(2):108-112, 1975.

4309 DEGLUTITION (CONTINUED): VI. MERCYISM *(Fr.)* Nemours-Auguste, S.; Barag, N. (Paul-Brousse Hosp., Villejiuf, France). *Ouest Med.* 27(17):1533-1540, 1974.

4310 DEGLUTITION (CONTINUED): VI. NEUROVEGETATIVE DYSKINETIC OR SPASMODIC DISORDERS. *(Fr.)* Nemours-Auguste, S.; Barag, N. (Paul-Brousse Hosp., Villejuif, France). *Ouest Med.* 27(21):1873-1896, 1974.

4311 A YEAST-LIKE INFECTION OF THE ESOPHAGUS CAUSED BY Lactobacillus acidophilus. *(E.)* McManus, J. P. A.; Webb, J. N. (Western Gen. Hosp., Univ. Edinburgh, Scotland). *Gastroenterology* 68(3): 583-586, 1975.

4312 GASTROESOPHAGEAL PROLAPSE. *(Ger.)* Miller, G. (no affil.). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):163-164, 1974.

4313 Candida albicans ULCER WITHIN HIATUS HERNIA SAC PRESENTING AS AN ULCERATED MASS. *(E.)* Binder, R. J.; Nelson, J. A. (VA Hosp., San Francisco, Calif.). *Gastroenterology* 68(3):587-590, 1975.

4314 INVESTIGATIONS ON THE REGULATION OF VASCULAR AND MUSCULAR FUNCTION IN THE TERMINAL ESOPHAGUS. *(Ger.)* Stelzner, F.; Kunath, U.; Kreuzer, J. (Cent. Surg., Univ. Frankfurt/Main, Germany). *Langenbecks Arch. Chir.* 336(4):303-321, 1974.

4315 HIATUS HERNIAS, SPHINCTER INCOMPETENCE, AND REFLUX ESOPHAGITIS: RADIOLOGICAL DIAGNOSIS. *(Ger.)* Frommhold, W. (Med. Inst. Radiol., Univ. Tubingen, Germany). *Langenbecks Arch. Chir.* 337(Suppl.):95-102, 1974.

4316 HIATUS HERNIA, SPHINCTER INCOMPETENCE, AND REFLUX ESOPHAGITIS: APPLICATIONS OF ENDOSCOPY, MANOMETRY, AND CONSERVATIVE THERAPY. *(Ger.)* Wienbeck, M. (2nd Med. Clin., Univ. Dusseldorf, Germany). *Langenbecks Arch. Chir.* 337(Suppl.):103-108, 1974.

4317 CLINICAL MANIFESTATIONS AND RADIOLOGICAL DIAGNOSIS OF ESOPHAGEAL-GASTRIC PASSAGE INSUFFICIENCY. *(Rus.)* Paltanavichius, K. I. (Vilna Dist. Hosp., Baltic Railroad, USSR). *Klin. Med. (Mosk.)* 52(7):72-74, 1974.

4318 PATHOPHYSIOLOGY OF THE LOWER ESOPHAGEAL SPHINCTER. *(Ger.)* Siewert, R. (Clin. Gen. Surg., Univ. Gottingen, Germany). *Langenbecks Arch. Chir.* 337(Suppl.):87-93, 1974.

4319 CRICOPHARYNGEAL MYOTOMY: TREATMENT OF DYSPHAGIA. *(E.)* Mitchell, R. L.; Armanini, G. B. (Dep. Surg., Stanford Univ., Calif.). *Ann. Surg.* 181(3):262-266, 1975.

4320 GASTRIC OESOPHAGOPLASTY IN THE TREATMENT OF OESOPHAGEAL ATRESIA. *(E.)* Drainer, I. K. (R. Hosp. Sick Child., Glasgow, Scotland). *J. R. Coll. Surg. Edinb.* 19(5):282-285, 1974.

4321 FAMILIAL INFANTILE ACHALASIA IN THREE SIBLINGS. *(Ger.)* Koteles, G.; Kemeny, P.; Reich, K. (Madarasz Hosp., Budapest, Hungary). *Monatschr. Kinderheilkd.* 123(1):9-14, 1975.

4322 THE POSSIBILITIES FOR OBJECTIFICATION OF THE RESULTS OF THERAPY OF ESOPHAGEAL ACHALASIA. *(Cz.)* Vaneckova, O.; Kazil, P.; Horacek, F.; Lukasova, A. (Surg. Clin. III, Prague, Czechoslovakia). *Rozhl. Chir.* 53(9):580-584, 1974.

4323 THE EFFECT OF OBESITY ON PATHOGENESIS OF HIATUS HERNIA. *(Pol.)* Miskiewicz, H. (Warsaw Acad. Med., Poland). *Wiad. Lek.* 27(6):501-505, 1974.

4324 ROENTGENOGRAPHS OF OPERATED HIATUS HERNIA IN EARLY INFANCY AND CHILDHOOD. *(Ger.)* Hormann, D. (Clin. Pediatr. Surg., Karl Marx Univ., Leipzig). *Radiol. Diagn. (Berl.)* 15(3):281-291, 1974.

4325 HIATUS HERNIAS: CLINICAL MANIFESTATIONS AND COMPLICATIONS. *(Ger.)* Koelsch, K. A. (Altstadt Reg. Hosp., Magdeburg, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):155-162, 1974.

4326 DIAGNOSIS AND TREATMENT OF DIAPHRAGMATIC HERNIAS. *(Rus.)* Glushkov, Iu. A. (Vladivostok Med. Inst., USSR). *Vestn. Khir.* (3):112-114, 1975.

4327 ESOPHAGEAL STRICTURE SECONDARY TO REFLUX ESOPHAGITIS. *(E.)* Safaie-Shirazi, S.; Zike, W. L.; Mason, E. E. (Univ. Iowa Coll. Med., Iowa City). *Arch. Surg.* 110(5):629-631, 1975.

4328 A GASTROPLASTY FOR SHORT ESOPHAGUS AND REFLUX ESOPHAGITIS: EXPERIMENTAL AND CLINICAL STUDIES. *(E.)* Demos, N. J.; Smith, N.; Williams, D. (New Jersey Med. Sch., Newark). *Ann. Surg.* 181(2):178-181, 1975.

4329 THE NONREFLUX DETERMINANT OF ESOPHAGITIS. *(E.)* Kalkay, M. N.; Cordoba, I.; Plevy, D. (East Orange VA Hosp., N.J.). *Am. J. Gastroenterol.* 63(2):135-146, 1975.

4330 TREATMENT AND COMPLICATIONS OF REFLUX OESOPHAGITIS. *(E.)* von der Oelsnitz, G.; Rehbein, F. (Bremen City Hosp., Germany). *Prog. Pediatr. Surg.* 7:57-68, 1974.

4331 EPIPHRENIC DIVERTICULUM IN ASSOCIATION WITH AN UNUSUAL MOTILITY DISTURBANCE: REPORT OF SURGICAL CORRECTION. *(E.)* Hurwitz, A. L.; Way, L. W.; Haddad, J. K. (Fort Miley VA Hosp., San Francisco, Calif.). *Gastroenterology* 68(4): 795-798, 1975.

4332 INTRAMURAL MICRODIVERTICULOSIS OF THE ESOPHAGUS. *(Ger.)* Thurnherr, N.; Faust, H.; Kapp, F.; Mihatsch, M. J. (Dep. Intern. Med., Univ. Basel, Switzerland). *Z. Gastroenterol.* 12 (8):603-604, 1974.

4333 DIFFUSE INTRAMURAL ESOPHAGEAL DIVERTICULOSIS. *(E.)* Graham, D. Y.; Goyal, R. K.; Sparkman, J.; Cagan, M. E.; Pogonowska, M. J. (Baylor Coll. Med., Houston, Tex.). *Gastroenterology* 68(4):781-785, 1975.

4334 OESOPHAGOGASTROSTOMY BYPASS-OPERATION AS AN ALTERNATIVE TO RESECTION FOR UNDILATABLE PEPTIC OESOPHAGEAL STENOSES. *(E.)* Hecker, W. C.; Hopner, F. (Univ. Child. Hosp., Munich, Germany). *Prog. Pediatr. Surg.* 7:69-81, 1974.

4335 THE PEPTIC OESOPHAGEAL STENOSIS. *(E.)* von Ekesparre, W. (Walddorfer Child. Hosp., Hamburg-Duvenstedt, Germany). *Prog. Pediatr. Surg.* 7:47-55, 1974.

4336 STENOSIS OF THE OESOPHAGUS SECONDARY TO HYPOPHARYNGEAL ABSCESS IN THE NEWBORN. *(E.)* Fritz-Mikulska, V. (Univ. Matern. Hosp., Zurich, Switzerland). *Prog. Pediatr. Surg.* 7:1-25, 1974.

4337 AIR IN THE ESOPHAGUS AS A SIGN OF SCLERODERMA (DIFFERENTIAL DIAGNOSIS WITH SOME OTHER ENTITIES). *(E.)* Martinez, L. O. (Mt. Sinai Hosp., Miami Beach, Fla.). *J. Can. Assoc. Radiol.* 25(3):234-237, 1974.

4338 CLINICAL AND RADIOLOGICAL STUDY OF THE ESOPHAGUS IN DIFFERENT STAGES OF SYSTEMIC SCLERODERMA. *(Rus.)* Savarovskii, A. I.; Mazneva, L. M.; Guseva, N. G. (Inst. Rheumatol., Moscow, USSR). *Sov. Med.* (7):103-108, 1974.

4339 ESOPHAGEAL FUNCTION IN SCLERODERMA. *(Ger.)* Krejs, G. J.; Lobsiger, M. M.; Rau, R.; Bron, B. A.; von Buren, U. S.; Pirozynski, W.; Blum, A. L. (Triemli Munic. Hosp., Zurich, Switzerland). *Z. Gastroenterol.* 12(Suppl.):412-413, 1974.

4340 THE RADIOLOGIST IN PREVENTION AND DIAGNOSIS OF INSTRUMENTAL PERFORATION OF THE ESOPHAGUS. *(E.)* Sullivan, M.; Berry, B. E.; Ferrante, W. A. (Ochsner Clin., New Orleans, La.). *South. Med. J.* 67(7):830-836, 1974.

4341 THE TREATMENT OF ESOPHAGEAL PERFORATION AND SO-CALLED SPONTANEOUS RUPTURE OF THE ESOPHAGUS. *(Ger.)* Scharfetter, H.; Bernard, W.; Weimann, S. (Surg. Clin., Univ. Innsbruck, Austria). *Zentralbl. Chir.* 99(44):1395-1401, 1974.

4342 SPONTANEOUS RUPTURE OF THE OESPHAGUS. *(E.)* Lawson, R. A. M.; Butchart, E. G.; Soriano, A.; Blesovsky, A.; Barnsley, W. (Newcastle Reg. Thorac. Cent., England). *J. R. Coll. Surg. Edinb.* 19(6):363-369, 1974.

4343 HAMARTOMATOUS POLYP OF THE ESOPHAGUS. *(E.)* Shah, B.; Unger, L.; Heimlich, H. J. (Jew. Hosp., Cincinnati, Ohio). *Arch. Surg.* 110(3): 326-328, 1975.

4344 A MELANOCYTIC TUMOR OF THE ESOPHAGUS. *(E.)* Assor, D. (Ohio State Univ. Hosp., Columbus). *Cancer* 35(5):1438-1443, 1975.

4345 ADENOID CYSTIC CARCINOMA OF THE ESOPHAGUS. *(E.)* Mukada, T.; Sato, E. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 113 (3):257-262, 1974.

4346 REVIEW OF RECENT CONCEPTS AND RESULTS REGARDING ESOPHAGEAL CARCINOMAS. *(Gr.)* Stamoulas, O. (no affil.). *Galenus* 15(4):560-570, 1973.

4347 MEDIASTINITIS IN LESIONS OF THE ESOPHAGUS IN CHILDREN. *(Ser.)* Krejovic, B. (Med. Fac., Univ. Belgrade, Yugoslavia). *Srp. Arh. Celok. Lek.* 102(1):45-50, 1974.

4348 RADIOLOGICAL DIAGNOSIS OF RECURRENT ESOPHAGEAL CANCER FOLLOWING RADIOTHERAPY AND COMBINED CHEMO- AND RADIOTHERAPY. *(Rus.)* Turganbaev, A. (Kazakh Res. Inst. Oncol. Radiol., Alma-Ata, USSR). *Med. Radiol. (Mosk.)* 19(12):13-16, 1974.

4349 CARCINOSARCOMA OF THE OESOPHAGUS (EPIDERMOID CARCINOMA WITH SARCOMA). *(Fr.)* Pelouin, A. B.; Vauclair, R. (Notre-Dame Hosp., Montreal, Canada). *Rev. Fr. Gastroenterol.* (97):35-42, 1974.

4350 CARCINOID SYNDROME IN A PATIENT WITH PRIMARY ESOPHAGEAL TUMOR. *(Ger.)* Broicher, K.; Hienz, H. A. (Munic. Hosp., Krefold, Germany). *Z. Gastroenterol.* 12(Suppl.):377-384, 1974.

4351 THE DIAGNOSIS OF AND PROGNOSIS FOR ESOPHAGEAL CARCINOMA. *(Ger.)* Wittman, I.; Huoranszki, F.; Laszlo, P.; Nogradi, E.; Tooth, E. (Janos Hosp., Budapest, Hungary). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):165-168, 1974.

4352 EXPERIENCE IN THE TREATMENT OF ESOPHAGEAL CARCINOMA IN THE UPPER AND MIDDLE THORACIC SEGMENT. *(Ger.)* Nakayama, K.; Nakayama, M.; Kinoshita, Y. (Tokyo Women's Med. Coll., Japan). *Zentralbl. Chir.* 99(43):1352-1357, 1974.

4353 THE TREATMENT OF ESOPHAGEAL CARCINOMA: A REPORT ON 267 CASES. *(Ger.)* Viikari, S. J.; Havia, T.; Inberg, M. V.; Scheinin, T. M. (Surg. Dep. Univ. Turku, Finland). *Zentralbl. Chir.* 99(43): 1358-1367, 1974.

4354 DIAGNOSTIC METHODS FOR THE DETECTION OF ESOPHAGEAL CARCINOMA. *(Ger.)* Berndt, H. (Cent. Inst. Cancer Res., Berlin, Germany). *Zentralbl. Chir.* 99(43):1345-1351, 1974.

4355 THREE DIFFERENT RADIOTHERAPY TECHNIQUES FOR ESOPHAGEAL CARCINOMA. *(Ger.)* Eichhorn, H. J.; Lessel, A. (Cent. Inst. Cancer Res., Berlin, Germany). *Zentralbl. Chir.* 99(49):1544-1557, 1974.

4356 REFLUX ESOPHAGITIS REFRACTORY TO MEDICAL MANAGEMENT. *(E.)* Custer, H. R. (no affil.). *Abdom. Surg.* 17(6):145-153, 1975.

4357 POSTOPERATIVE CHYLOTHORAX. SIX CASES IN 2,500 OPERATIONS, WITH A SURVEY OF THE WORLD LITERATURE. *(E.)* Cevese, P. G.; Vecchioni, R.; D'Amico, D. F.; Cordiano, C.; Biasiato, R.; Favia, G.; Farello, G. A. (Sch. Med., Univ. Padua, Italy). *J. Thorac. Cardiovasc. Surg.* 69(6):966-971, 1975.

4358 CONGENITAL ESOPHAGEAL STENOSIS WITH LACK OF THE SUBMUCOSA. *(E.)* Takayanagi, K.; Ii, K.; Komi, N. (Tokushima Univ. Hosp., Japan). *J. Pediatr. Surg.* 10(3):425-426, 1975.

4359 PSEUDOTUMORAL VARICES OF ESOPHAGUS. *(E.)* Donner, D.; Esquerre, F.; Smulewicz, J. J. (Queens Hosp. Cent., N.Y.). *N.Y. State J. Med.* 75(6):898-899, 1975.

4360 ESOPHAGEAL CARCINOMA ARISING ON BARRETT'S EPITHELIUM. *(E.)* Alves, M. A.; Ellis, F. H.; Haggitt, R.; Colcher, H. (Lahey Clin. Found., Boston, Mass.). *Gastrointest. Endosc.* 21(4):183, 1975.

4361 ACHALASIA: A RETROSPECTIVE CORRELATION. *(E.)* Biederman, M. A.; Rinaldo, J. A.; Galzayd, E. (Providence Hosp., Southfield, Mich.). *Gastrointest. Endosc.* 21(4):183-184, 1975.

4362 STRANGULATED DIAPHRAGMATIC HERNIA. A CLINICAL STUDY. *(E.)* Christiansen, L. A.; Blichert-Toft, M.; Bertelsen, S. (Rigshosp., Copenhagen, Denmark). *Am. J. Surg.* 129(5):574-578, 1975.

4363 HIATAL HERNIA AND GASTROESOPHAGEAL REFLUX IN INFANCY AND CHILDHOOD. ANALYSIS OF THE RADIOLOGIC FINDINGS. *(E.)* Darling, D. B. (Boston Floating Hosp. Infants Child., Mass.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4):724-736, 1975.

4364 INFLAMMATORY PSEUDOTUMORS (INFLAMMATORY FIBROUS POLYPS) OF THE ESOPHAGUS. A CLINICOPATHOLOGIC STUDY. *(E.)* LiVolsi, V. A.; Perzin, K. H. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Am. J. Dig. Dis.* 20(5):475-481, 1975.

ESOPHAGUS

4365 PROLAPSE OF THE GASTROSTOMY CATHETER IN CHILDREN. *(E.)* Currarino, G.; Votteler, T. (Child. Med. Cent., Dallas, Tex.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4):737-741, 1975.

See also, 4187, 4195, 4200, 4211, 4216, 4217, 4218, 4421, 4447, 4486, 5054, 5062, 5089, 5094, 5126, 5185.

STOMACH

4366 GASTRIC CONSEQUENCES OF LONGTERM HYPERCAPNIA IN THE RAT. *(Fr.)* Reichart, E.; Guerrin, F.; Gosselin, B. (Fac. Med., Lille, France). *Biomedicine* 20(5):366-373, 1974.

Continuous hypercapnia without hypoxia was induced in 78 Wistar rats by allowing them to breathe a mixture containing 8 or 10% CO_2 for 1-6 weeks. Some rats were also subjected to a mild hypoxia (17-18%) for 3 days and some had the hypercapnia interrupted for 1 or 7 days before they were sacrificed . Gross and microscopic examinations of the stomachs showed changes in the gastric mucosa in 53/56 rats subjected to hypercapnia for 15 days. Abnormalities included catarrhal or hemorrhagic gastritis, frequent and large areas of desquamation, erosions, and a few ulcerations. Hypercapnia of more than 15 days duration increased desquamation and produced superficial retraction of the mucosa. Hypoxia only mildly aggravated the symptoms, and when the hypercapnia had been discontinued 7 days prior to sacrifice, most lesions had disappeared. It is suggested that local changes in the microcirculation cause most of the gastric lesions, and these results may help clarify gastrointestinal complications which may occur during respiratory insufficiency in man.

4367 HISTOCHEMICAL STUDIES OF ACID PHOSPHATASE IN TUMOR CELLS OF GASTRIC CARCINOMA. *(Rus.)* Dikshtein, E. A.; Vasilenko, I. V. (Donetsk Med. Inst., USSR). *Arkh. Patol.* 36(11):67-72, 1974.

Histochemical studies of the activity of acid phosphatase with β-glycerophosphate at pH 6.0 in 40 gastric tumors (17 adenocarcinomas, six solid cancers, 17 undifferentiated polymorphocellular and mucous cancers) revealed three groups of tumors: with weak (12), moderate (13) and high (15) activity of the enzyme. Phosphatase activity was correlated with the content of fat, total protein, SH-group protein, RNA and amount of mucous secretion in the tumor cells. Undifferentiated tumors appeared to have higher acid phosphatase activity than adenocarcinomas. No direct correlation was found between acid phosphatase and total protein or SH-groups, while a high acid phosphatase activity was accompanied by a low RNA content in the cytoplasm of the tumor cells. It is suggested that acid phosphatase is involved in autolysis, but its involvement in secretion is less obvious; there is an inverse correlation between enzyme and protein synthesis. It is concluded that the activity of acid phosphatase is more indicative of early dystrophic changes in the tumor cells than the amount and distribution of fat.

4368 HISTOLOGICAL AND HISTOCHEMICAL FEATURES OF THE STROMA IN STOMACH CANCER. *(Rus.)* Zolotarevskii, V. B.; Vorobieva, A. A. (1st Moscow Med. Inst. USSR). *Arkh. Patol.* 36(11):61-67, 1974.

Studies were performed on 60 resected stomachs with adenocarcinoma (15), solid cancer (18) and glandular (6), scirrhus (9), undifferentiated (10), and mixed (2) tumors. Three types of stroma were identified. (1) Vascular stroma, found in undifferentiated tumors and adenocarcinomas, consisted of narrow interlayers with small lymph and blood vessels surrounded by a few argyrophilic and collagen fibers and a small number of fibroblasts. No deposits of acid or neutral mucopolysaccharides (MPS) were observed. (2) Granulation tissue-type stroma was identified in all histological types of cancer. It is generally densely infiltrated with leukocytes, plasma cells, histiocytes and lymphoid cells. Acid MPS are either nonexistant or are present in very small amounts. The connective tissue and fibers of the stroma were indicative of immaturity and precollagen and collagen fiber formation. (3) Fibrous stroma was observed in adenocarcinoma and solid and scirrhus tumors. Most collagen fibers stained readily with fuchsin. Fibrous stroma always contained neutral and acid MPS in the interlayers where active synthesis of collagen occurs. The findings suggest a correlation between the presence of fat cells near the tumor and the accumulation of acid MPS in the stroma. Fibroblast proliferation and the increased MPS content in parts of the stomach near the tumor suggest the stroma develops before infiltration of cancer cells.

4369 GASTRIC ULCERATION IN THE PIG SUBJECTED TO HAEMORRHAGIC SHOCK. *(E.)* Kivilaakso, E.; Kalima, T. V.; Lempinen, M. (Second Dep. Surg., Univ. Helsinki, Finland). *Scand. J. Gastroenterol.* 9(7):685-690, 1974.

The mechanism of ulcerogenesis and the roles of transient mucosal ischemia, gastric reflux of bile, and disruption of the mucosal barrier to the backdiffusion of H^+ in the pathogenesis of stress ulcer were studied in 17 piglets. The fasted piglets were subjected to hemorrhagic shock produced by bleeding from a cannulated femoral artery. The gastric motility and the development of the mucosal changes during the shock period and shortly afterwards were followed by endoscopy. Mucosal lesions developed in all but 1 of the 12 piglets which survived the 3 hr of hemorrhagic shock. Endoscopic examination during exsanguination showed inhibition,

followed by complete cessation of gastric motilin; this in turn was followed by a copious reflux of bile and the duodenal contents into the stomach. The first mucosal changes, seen soon after the establishment of shock, comprised a network of ischemic and hyperemic areas with extravasation of blood. Toward the end of the shock period, small mucosal hematomas developed within these areas. After transfusion of blood through a femoral vein, these areas became intensely red-colored with transient focal hemorrhages and, later, small superficial erosions were seen. More extensive lesions, confined almost exclusively to the body of the stomach, were seen 24 hr after the cessation of shock; these lesions included 'punched-out' ulcers, superficial ulcerations, and areas of hemorrhagic gastritis. The results indicate that the bleeding shock model is a reproducible and reliable method of inducing stress ulcers in piglets. They also suggest that capillary injury, transient mucosal ischemia, and the gastric reflux of bile and/or the duodenal contents are important factors in the genesis of gastric stress ulcers.

4370 DOUBLE-BLIND STUDY ON THE TREATMENT OF GASTRIC ULCER WITH CARBENOXALONE. *(Fl.)* van Camp, F.; Standaert, L. (St. Elisabeth Hosp., Antwerp, Belgium). *Tijdschr. Gastroenterol.* 17(1):51-63, 1974.

A double-blind trial of carbenoxalone was performed on 36 patients with gastric ulcers. Patients with hypertension, cardiac decompensation, and renal damage were excluded, as were four patients who were later found to have stomach cancer, one whose treatment had to be discontinued because of acute appendicitis, and another who stopped taking his medicine. Of the remaining 30 patients, 13 had primary gastric ulcers while 17 were being treated for secondary ulcers or recurrences. This trial was performed on 21 hospitalized patients and six outpatients; three were treated as both inpatients and outpatients. Carbenoxalone was administered to 14 patients in doses larger than those usually recommended: 400 mg/day for the first 2 weeks and 200 mg/day for the next 4 weeks; controls consisted of 16 patients who received placebos. Complete disappearance of the ulcer or a scar with an area of 2 mm^2 at most, as seen on radiological or gastroscopic examination, was observed in eight (66%) of carbenoxalone-treated patients and six (43%) of the controls; improvement occurred in one (8%) and four (29%), resp.; no change was observed in three (25%) and three (21%), resp.; and the condition of one of the controls (7%) became worse. After 3 months of carbenoxalone therapy, 10 (83%) of the carbenoxalone-treated patients were completely recovered. After 6 weeks of therapy, no appreciable difference in weight gain was observed between the two groups, but there was a clearly increased tendency for carbenoxalone-treated patients to retain water and develop edema. It was not clear whether increased ingestion of milk or the presence of sodium bicarbonate in antacids might have contributed to the edema. Reversible increases in blood pressure occurred in two treated patients. Serum sodium concentrations increased in nine (63%) of the treated patients and in six (37%) of the controls, while serum potassium levels decreased in 10 (70%) of the treated patients and in five (36%) of the controls. These changes in serum electrolytes are attributed to water retention which was most pronounced in patients who responded the most rapidly to carbenoxalone. Increases in serum transaminase values, which occurred primarily during week 2 or 3 of treatment, occurred in 12 treated patients and in only two controls; these values later returned to their initial levels.

4371 ENDOSCOPIC FOLLOW-UP OF GASTRIC POLYPS. *(E.)* Mizuno, H.; Kobayashi, S.; Kasugai, T. (Aichi Cancer Cent. Hosp., Nagoya, Japan). *Gastrointest. Endosc.* 21(3):112-115, 1975.

The course and malignant changes in a series of 118 patients with gastric polyps (benign) were followed over a period of 1-6.5 yr using endoscopy and histopathological techniques. Of these cases, 114 remained benign and 4 showed malignancy in the final histologic examination. Endoscopic findings in the benign group (61 men and 53 women, average age 54.2 yr) indicated that the rate of morphological change increased with increasing period of observation. The polyps tended to grow over a period of several yr and thereafter remain at about the same size (within 2 cm); in some patients a similar, but reverse, trend was observed. Among the malignant group was a 50-year-old man with a sessile polyp and a pedunculated polyp. The latter increased moderately in size with gradual lobulation; the histological picture suggested an adenocarcinoma engrafted on a polypoid adenoma. In the remaining patients (62- and 64-year-old men and a 63-year-old woman), 2 sessile polyps and a polyp of the lower body of the stomach eventually revealed adenocarcinoma. The results suggest that endoscopic follow-up of benign gastric polyps is justified in that malignant change may occur in a small number of cases.

4372 THE USE OF METOCLOPRAMIDE IN ACUTE PYLORIC OBSTRUCTION: A CLINICAL TRIAL. *(E.)* Zer, M.; Dintsman, M. (Beilinson Hosp., Petah Tiqva, Israel). *Am. J. Gastroenterol.* 63(3):232-239, 1975.

Twelve patients (8 men and 4 women, aged 14-68 yr) with complete or partial pyloric obstruction were treated with metoclopramide in an attempt to improve their preoperative state, facilitate radiography, and permit elective surgery. All cases presented with repeated intractable vomiting and would have required urgent surgery. After initial treatment by gastric decompression, i.v. fluids, and electrolyte replacement, 10 mg metoclopramide were administered i.v. or i.m. 3 times daily. Drainage from the gastric tube was measured. After 24 hr an attempt was made to close the gastric tube, withdraw it, and begin p.o. feeding. The drug was continued with a p.o. dose of 30-60 mg/day. If tube removal was unsuccessful, the drug dose was doubled. If this failed to improve the patient's condition, surgery was performed as soon as possible. Of the 12 patients, 8 (66.6%) had very good results; vomiting stopped and p.o. feeding was resumed. Six later had surgery, and diagnosis of duodenal ulcer with severe pyloric stenosis was confirmed. Two cases (16.6%) had good results; vom-

iting stopped, but severe epigastric discomfort continued, preventing p.o. feeding. In the final two cases, results were poor; the attempts to withdraw the gastric tube failed and semiemergency surgery was performed. Severe scarring and complete pyloric obstruction were found. It is concluded that metoclopramide may effectively improve gastric emptying, sometimes even in complete pyloric obstruction, and allow patients better preparation for surgery.

4373 ROENTGEN PROBLEMS IN EVALUATING THE ATROPHIC STOMACH OF THE ELDERLY. *(E.)* Bryk, D.; Elguezabai, A. (Jewish Hosp. Med. Cent. Brooklyn, N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):236-241, 1975.

Radiological simulation by gastric atrophy of other conditions was reviewed in 17 patients, 65-89 yr. In eight cases, scirrhous carcinoma of the distal stomach was simulated. Radiograms showed limited distensibility of the stomach with straightening of the lesser and indentations of the greater curvature. Peristalsis was absent or minimal. In two cases there was a scalloped, coarse irregularity of the distal greater curvature. Biopsies in four cases showed absence of malignancy. In six of the 17 cases, gastric retention of food after an overnight fast falsely indicated gastric outlet obstruction. Barium study in these cases showed normal peristalsis with no delay of pyloric opening or gastric emptying. The usual features of gastric atrophy were observed. In three of the cases, there was gastric retention of p.o. cholecystographic medium 14 hr after ingestion. Again, roentgen studies showed features of gastric atrophy, but no delay in pyloric opening or gastric emptying. It is suggested that physicians be aware of such problems in roentgen diagnosis.

4374 EFFECT OF NAPROXEN ON GASTROINTESTINAL MICROBLEEDING FOLLOWING ACETYLSALICYLATE MEDICATION. *(E.)* Arsenault, A.; Varady, J.; LeBel, E.; Lussier, A. (Cent. Hosp. Univ., Sherbrooke, Quebec, Canada). *J. Clin. Pharmacol.* 15(4):340-346, 1975.

Naproxen was substituted for acetylsalicylic acid to determine its influence on salicylate-induced gastrointestinal bleeding. Normal volunteers (12) were given increasing doses of salicylate until guaiac tests were positive. Venous blood (16 ml) was drawn, incubated with 200 µCi sodium chromate 51, and the labeled erythrocytes washed and reinjected into the subjects. For the first two weeks of the study, subjects were continued on 2.7-4.5 g/day of acetylsalicylic acid. For the last 2 weeks, the acetylsalicylic acid was stopped; six subjects were given 250 mg naproxen twice daily, and 6 were given a placebo in a double-blind design. Stools were collected on the last three days of each week, and 5 ml of blood was drawn at the beginning and end of each collection period. All samples were counted in a well-type gamma counter. Blood in the stools was quantitated by dividing the net counts/min in the total specimen by the net counts/min/ml blood. The placebo group showed a clear-cut drop in blood loss. A two-way analysis of variance showed a highly significant non-additivity of the Tukey type. On a natural logarithmic scale, the only significant contrast was weeks on acetylsalicylic acid *versus* weeks on placebo therapy. In the naproxen group, there was also a clear-cut difference between weeks on acetylsalicylic acid and weeks on naproxen. Two-way variance analysis showed significant non-additivity. Analysis on a logarithmic scale showed a highly significant contrast between microbleeding during acetylsalicylic acid and naproxen treatment. There was no consistency between acetylsalicylic acid dose and level of microbleeding in either the naproxen or placebo groups. It is concluded that substitution of naproxen for acetylsalicylic acid will significantly reduce microbleeding to near normal levels.

4375 GASTRIC MUCOSAL MORPHOLOGY IN TROPICS AND INFLUENCE OF SPICES, TEA, AND SMOKING. *(E.)* Tyagi, K. P.; Mukhopadhyay, A. K.; Agarwal, H. N.; Naik, S. R.; Malik, G. B.; Gupta, D. N.; Chuttani, H. K. (Maulana Azad Med. Coll., New Delhi, India). *Nutr. Metab.* 17(3):129-135, 1974.

Gastric mucosal morphology in the tropics was studied to compare the frequency of gastritis in normal and dyspeptic subjects, and to determine the effects of spices, tea, and tobacco. The dyspeptic group consisted of 82 patients with symptoms suggestive of gastritis, while 6% normal adults served as controls. Peroral gastric biopsies were done in all subjects as well as barium meal examinations. Subjects received either a calculated diet or diet was determined by questionnaire. The amount of spices consumed was calculated by dividing the total amount consumed/family/month by the number of members/family. Spices included *Capsicum frutescens, Elettaria cardamomum, syzigium, armalicum, and Coriandrum sativum.* Tea and cigarette consumption was assessed by questionnaire. Changes of chronic gastritis were significantly higher in the patients (42 of 82) than in the controls (23 of 76). Both superficial and atrophic gastritis were more common in the patients; no subject, patient or control, had gastric atrophy. No radiological evidence of gastroduodenal ulceration was found in either group. Radiological grading of the folds showed 16 of 41 controls and 17 of 31 patients had abnormal folds. No significant differences were noted between moderate and heavy spice consumers in morphological changes of gastritis, or between moderate and heavy tea drinkers. There was no significant difference in occurrence of gastritis between smokers and non-smokers. It is concluded that spices, tea, and smoking are not correlated with occurrence of gastritis.

4376 ROLE OF TAUROCHOLIC ACID IN PRODUCTION OF GASTRIC MUCOSAL DAMAGE AFTER INGESTION OF ASPIRIN. *(E.)* Cochran, K. M.; Mackenzie, J. F.; Russell, R. I. (R. Infirm., Glasgow, Scotland). *Br. Med. J.* 1(5951):183-185, 1975.

The possibility that aspirin-induced gastric mucosal damage may occur more readily in the presence of

bile was studied in 22 healthy subjects. The subjects were fasted overnight and a nasogastric tube with a probe electrode attached was passed into the body of the stomach. The gastric transmucosal electrical potential difference (E.P.D.) was measured after the stomach had been emptied of any resting secretion, in the presence of 50 ml of normal saline, and in the presence of aspirin (600 mg suspended in 50 ml of normal saline) and/or taurocholic acid (TCA, 5 mM/liter in normal saline solution). After the introduction of acetylsalicylic acid in 7 subjects the mean E.P.D. fell significantly, from a baseline value of -33.3 mV to -17 mV. After the introduction of TCA in 7 additional subjects, the mean E.P.D. again fell significantly, from a baseline value of -38.1 mV to -19.1 mV. The mean duration of the changes following aspirin and TCA administration was 14.4 and 17.5 min, resp. When 8 additional subjects were administered a combination of aspirin and TCA, the mean E.P.D. fell from a baseline value of -38.6 mV to -17.9 mV, but the mean duration of this change (27 min) was significantly longer than that after either agent alone. These results indicate that the ingestion of aspirin, together with coincidental reflux of bile from the duodenum, may be a factor in the pathogenesis of aspirin-induced gastric mucosal damage.

4377 NARCOTIC ANALGESICS AND DELAYED GASTRIC EMPTYING DURING LABOUR. *(E.)* Nimmo, W. S.; Wilson, J.; Prescott, L. F. (R. Infirm., Edinburgh, Scotland). *Lancet* 1(7912):890-893, 1975.

The effects of pethidine, diamorphine (heroin), pentazocine, and metoclopramide on gastric emptying during labor were studied in 56 patients. The women were receiving pethidine (150 mg), diamorphine (10 mg), pentazocine (60 mg), and/or metoclopramide (10 mg) i.m. as needed. Paracetamol (acetaminophen, 1.5 g) was given p.o. at varying times in relation to the narcotic injections and the imminence of delivery. The rate of gastric emptying was measured indirectly based on the kinetics of absorption of the paracetamol. The absorption of paracetamol was also studied 2-5 days postpartum in 10 patients who had received narcotics during delivery. Paracetamol absorption was normal in the postpartum patients and in a control group during delivery. Pethidine and diamorphine significantly reduced the absorption of paracetamol as judged by the plasma-paracetamol concentrations 2 hr after ingestion and the time taken to reach maximum levels. Paracetamol absorption was also markedly delayed in the women taking pentazocine and metoclopramide; the latter drug did not significantly enhance the rate of paracetamol absorption in the patients taking pethidine or diamorphine. There was no relation between delayed paracetamol absorption and the parity of the patients or the administration of cyclizine or oxytocin. The results suggest that the use of pethidine, diamorphine, or pentazocine for the relief of pain in labor may be a major factor responsible for maternal death due to delayed gastric emptying; patients treated with narcotics will also be more likely to vomit or to have increased gastric contents, and they are unlikely to be helped by metoclopramide.

4378 GASTRIC SECRETION IN HYPERPARATHYROID SUBJECTS BEFORE AND AFTER SURGERY. *(E.)* Johansson, H.; Furberg, B.; Krause, U.; Lundqvist, G.; Rimsten, A. (Univ. Hosp., Uppsala, Sweden). *Ups. J. Med. Sci.* 79(3):155-160, 1974.

The effects of parathyroidectomy on the gastric secretion and plasma gastrin levels in patients with hyperparathyroidism were studied. Patients with histologically verified hyperparathyroidism (61) were studied; 41 were preoperatively hypercalcemic, while 20 appeared to be preoperatively normocalcemic. Symptoms of gastritis and/or peptic ulcer disease were present in 11 patients. The gastric secretion rate and hydrogen iqn concentration were measured after histamine-stimulation before and 4 weeks after parathyroidectomy. The serum gastrin content was measured by a solid-phase radioimmunoassay. In all patients, the serum calcium level decreased after surgery, the levels being independent of the presence or absence of peptic ulcer disease. Achylia after submaximal histamine stimulation was found in one normocalcemic patient and six hypercalcemic patients, and hypochylia was found in a similar number of patients. The gastric output did not differ significantly before and after surgery in either group, and the preoperative values were of the same magnitude in the two groups. The sialic acid content in the gastric juice also did not change after surgery, but the values were consistently lower in the normocalcemic patients than in the hypercalcemic patients. The serum gastrin level was not influenced by parathyroidectomy. The clinical results suggest that performing parathyroid surgery before surgical treatment of ulcer disease in hyperparathyroid patients with peptic ulcer may be of value.

4379 HEMORRHAGE DUE TO DIFFUSE EROSIVE GASTRITIS. *(E.)* Stremple, J. F.; Elliott, D. W. (Univ. Pittsburgh Sch. Med., Pa.). *Arch Surg* 110 (5):606-612, 1975.

Operative experience (5 yr) with bleeding erosive gastritis is reviewed to help define the safest and most effective surgical approach. Of 796 patients admitted with upper gastrointestinal bleeding during this period, 156 had erosive gastritis. Vigorous nonoperative therapy stopped the bleeding in 117 cases. This treatment program consisted of iced saline lavage, transfusion with packed red blood cells, platelets, or whole blood, vitamin K administration where indicated, antacid administration *via* gastric tube, parenteral anticholinergic therapy, and endoscopic examination. In 39 cases, emergency operations were performed to control hemorrhage. In 24 of these patients (group A), subtotal gastric resection with truncal vagotomy was performed, and in the remaining 15 patients (group B), the operation was limited to pyloroplasty, truncal vagotomy, and multiple suture ligation. The group B patients were, on the average, 20 yr older than the group A patients, and 12 had life-threatening preoperative problems as compared with only four patients in group A. The operative mortality in group A was 42%, with recurrent bleeding occurring in 33%. In group B, only 17% showed recurrent bleeding and the operative mortality was 45%. More than half the deaths in

group B were due to sepsis which was present preoperatively. Rebleeding was the cause of death in 33.3% of the group A fatalities and in none of the group B fatalities. The results indicate that truncal vagotomy with pyloroplasty and multiple suture ligation is a good choice of operation in the high-risk and elderly patient in whom the main issue is immediate salvage of life in the face of potential exsanguination.

4380 GASTRIC MUCOSAL CHANGES IN IDIOPATHIC AND POST-HAEMORRHAGIC IRON DEFICIENCY ANAEMIA - A COMPARITIVE STUDY. *(E.)* Gupta, S. P.; Arya, R. K. (Med. Coll., Rohtak, India). *Indian J. Med. Res.* 62(11):1751-1756, 1974.

Gastric morphological and acid-secretory patterns were studied in 20 cases of idiopathic iron deficiency anemia (Group A) and compared with those observed in age- and sex-matched cases of post-hemorrhagic iron deficiency anemia (Group B). The histamine-stimulated acid output was determined and mucosal changes were studied in gastric biopsy specimens. Gastric musocal abnormalities were present in 4/5 of the patients in both groups: atropic gastritis was slightly more common in group A and superficial gastritis was slightly more common in group B. Histamine-fast achlorhydria was present in seven males and two females in group A and six females in group B. The prevalence of gastric abnormalities did not vary significantly with age or sex in either group, although atrophic gastritis was significantly more common in women than in men. The results suggest that the nature of the association between iron deficiency and gastric mucosal lesions is similar in the post-hemorrhagic and idiopathic types of iron deficiency anemia, but that the factors responsible for the negative iron balance are overt in the formen and more subtle in the latter condition. A greater chronicity of iron deficiency may be associated with more severe and permanent gastric changes.

4381 AN ENDOSCOPIC EVALUATION OF THE EFFECTS OF NON-STEROIDAL ANTI-INFLAMMATORY DRUGS ON THE GASTRIC MUCOSA. *(E.)* Lanza, F.; Royer, G.; Nelson, R. (Baylor Coll. Med., Houston, Tex.). *Gastrointest. Endosc.* 21(3):103-105, 1975.

The effects of placebo (lactose, q.i.d.), phenylbutazone (100 mg, q.i.d.), aspirin (900 mg, q.i.d.). indomethacin (25 mg, q.i.d.), and ibuprofen (a new, nonsteroidal antiinflammatory agent) (300 mg, q.i.d.) on the gastric mucosa were studied in a prospective, single-blind crossover study on 20 normal volunteers. All drugs were given for 7 days. Stool were examined periodically by the guaiac method and the gastric mucosa was evaluated directly before and after drug treatment by endoscopy at the time of gastroscopy. Varying degrees of erythrema, edema, petechial hemorrhage, and gastric erosions were seen with all 4 drugs, the most severe abnormalities being observed with aspirin and indomethacin. The abnormalities caused by ibuprofen were significantly less severe than those induced by aspirin and indomethacin. With aspirin and indomethacin, the degree of mucosal injury was more severe than the subjective symptoms recorded by the subjects; with phenylbutazone, there was almost a direct linear correlation between symptoms and endoscopic findings; and with ibuprofen, there was little mucosal abnormality and minimal subjective symptoms. All drugs were absorbed to a significant extent, and none caused gastrointestinal bleeding, blood chemistry aberrations, or hematologic changes. The findings support previous data indicating minimal gastric mucosal injury with ibuprofen. They also suggest that the cause of sudden painless gastrointestinal bleeding in patients taking antiinflammatory drugs is acute hemorrhagic gastritis.

4382 HEMORRHAGIC EROSIVE GASTRITIS: A CLINICAL STUDY. *(E.)* Lee, E. R.; Dagradi, A. E. (Kaiser-Permanente Bellflower Med. Cent., Calif.). *Am. J. Gastroenterol.* 63(3):201-208, 1975.

The incidence of hemorrhagic erosive gastritis was studied in 400 consecutive patients from a civilian general hospital (over a 7-yr period) presenting with upper gastrointestinal hemorrhage. Age and sex distribution was representative of the general population; race and socio-economic status was a cross section of Southern California. All were examined by endoscopy. Hemorrhagic erosive gastritis was found in 74 of the patients (18.5%), of whom 53 were male and 21 female. This occurred despite a preponderance of female patients (61.8% female versus 38.2% male over a 1-yr period) at the hospital. Of the 74, 55 (74%) had a history of alcohol or salicylate intake. In relating sex distribution to etiologic agent, males were predominant, but no appreciable age difference existed between the sexes in relation to the etiologic agent. Additional peptic lesions were found in 32 patients (43%) and gastric ulcers in 15. In 45 of 54 patients, inflammatory mucosal disease was present, and five had hypertrophic hypersecretory gastropathy. Eleven patients had previously undergone surgery for peptic ulcer. There was a 5% mortality; 2 patients died of hepatic failure, 1 from ruptured esophageal varices, and 1 from fulminant serum hepatitis. This study corroborates findings of a previous all-male VA hospital study. It is concluded that in hemorrhagic erosive gastritis there is a preponderance of males affected, alcohol and/or salicylate is the most common offending agent, and the disease is commonly associated with other gastric mucosal abnormalities.

4383 EARLY GASTRIC CANCER - EXPERIENCE IN GERMANY. *(E.)* Elster, K.; Kolaczek, F.; Shimamoto, K.; Freitag, H. (Munic. Hosp., Bayreuth, Germany). *Endoscopy* 7(1):5-10, 1975.

Cases of early gastric cancer (87) were analyzed and a technique described which allows easy correlation between histological pattern and gross observation. Cases from 1969-1973 were studied; the term early cancer marked not only the spread of invasion but a diagnosis made early in life span. The differentiation between mucosal and submucosal invasion was found to be of special importance in regard to prog-

nostic evaluation of early gastric cancer. Signet ring cell carcinoma was more common in younger age; highly differentiated adenocarcinoma predominated in old age. In 69 of 87 lesions, the first biopsy confirmed the diagnosis of carcinoma. The data suggest that by intensifying gastroscopic biopsy the frequency of diagnosed early cancers will increase.

4384 BENEFICIAL EFFECT OF HUMAN GROWTH HORMONE ON STRESS ULCERS. *(E.)* Winawer, S. J.; Sherlock, P.; Sonenberg, M.; Vanamee, P. (Mem. Sloan-Kettering Cancer Cent., New York, N.Y.). *Arch. Intern. Med.* 135:569-572, 1975.

The potential value of human growth hormone in controlling hemorrhage from stress ulcers was evaluated in 8 patients who had undergone massive upper gastrointestinal tract hemorrhage from verified stress ulcers. Growth hormone was administered i.m. at 10 mg/kg/day for 6 days or until bleeding stopped. Untreated patients with stress ulcer hemorrhage served as controls. Human growth hormone was effective in healing erosions and controlling hemorrhage in 6 of the 8 stress ulcer patients; 2 other patients died during the study, 1 from sepsis and the other from cardiac arrest. Among the patients who responded to treatment, 3 required 4 days of hormone treatment, 2 required 6 days of treatment, and 1 required 18 days of treatment. In the control group, 6 of 8 patients died with continued bleeding. In the patients who responded to hormone treatment, blood urea nitrogen levels were consistently reduced, but the effects on blood sugar, phosphorus, and fibrinogen levels were inconsistent. Although these observations must be extended to additional stress ulcer patients in a larger randomized study, they support data from animal studies and suggest a possible role of human growth hormone in the treatment of human stress ulcers.

4385 PATHOPHYSIOLOGY OF STRESS ULCER AND ITS PREVENTION. I. PHARMACOLOGIC DOSES OF STEROID. *(E.)* Kawarda, Y.; Weiss, R.; Matsumoto, T. (Hahnemann Med. Coll. Hosp., Philadelphia, Pa.). *Am. J. Surg.* 129:249-254, 1975.

The efficacy of large doses of the dexamethasone in the prevention of stress ulcer formation in restrained rats was studied by microangiohistologic techniques. Fasted male Wistar rats were restrained and kept at 4 C for 2 hr. Some of the animals were pretreated (0.1 mg/kg/day for 7 days, or 10 mg/kg 24 hr before restraint) or posttreated (10 mg/kg immediately after restraint) with dexamethasone. Four hr after restraint, the animals were examined for ulcers. In a second study, a double-color perfusion technique was used to study the morphologic changes in the stomach 4 hr after restraint. Compared with the untreated controls, the incidence and severity of ulcers was significantly reduced in the rats pretreated with 10 mg/kg steroid 24 hr before restraint. The other pretreatment group and the posttreatment group did not differ significantly from the controls in terms of ulcer incidence or severity. The microfile perfusion study showed vasoconstriction and focal ischemia in the gastric mucosa of the control group, the posttreatment group, and the group pretreated with 0.1 mg/kg/day steroid for 7 days. Although small areas of ischemia were seen in the group pretreated with a 10 mg/kg dexamethasone, the findings in this group were similar to those in the normal stomach. The results indicate that a large dose of dexamethasone may protect against vasoconstriction, increase inotropic action under conditions of stress, or inhibit damage to gastric cell membranes when given prior to the development of a stress ulcer.

4386 ACUTE GASTRIC MUCOSAL DAMAGE INDUCED BY BILE SALTS, ACID, AND ISCHEMIA. *(E.)* Ritchie, Jr., W. P. (Univ. Virginia Med. Cent., Charlottesville). *Gastroenterology* 68(4):699-707, 1975.

The ulcerogenic potential of topically applied acid and bile salts, alone and in combination with vasopressin-induced mucosal ischemia, was studied. In two groups of healthy adult dogs, vascularized full thickness wedges of a gastric wall were mounted in lucite chambers, and, with mucosa directly visualized, were studied during three consecutive 30-min periods. Group A (five dogs) was subjected sequentially to: topical acid test solution (ATS); ATS plus vasopressin (VP; 0.1 U/kg/min); and ATS plus VP plus 5mM sodium taurocholate (TC). Group B (five dogs) was subjected to ATS, ATS plus TC, and ATS plus VP plus TC. Three groups of controls were also studied: group A^1 were subjected to ATS plus VP during periods 2 and 3; group B^1 was subjected to ATS plus TC during periods 2 and 3; and group C was subjected to topical "neutral" test solution (NTS, 160 mM NaCl), NTS plus TC, and NTS plus VP plus TC. During each period, the net fluxes in volume, H^+, and Na^+, the electrical potential difference, and theaminopyrine clearance, a measure of gastric mucosal blood flow, were determined. Mucosal damage was assessed by an independent observer using photographs obtained after each period. The results indicated that significant lesion formation occurred only in the presence of acid, bile salts, and ischemia, No lesions were noted during any period in the control animals. The data suggest that lesion formation was not a function of either back diffusion of H^+ or of mucosal ischemia, *per se*. Relative to period 1, mucosal blood flow was significantly increased in non-ischemic mucosa exposed to bile salts during vasopressin infusion. Perhaps as a consequence, the combination of mucosal ischemia and topically applied acid and bile salts is severely, acutely, and uniquely ulcerogenic.

4387 ENZYMOLOGICAL CHANGES IN ABNORMAL DIFFERENTIATION: INTESTINAL METAPLASIA IN HUMAN GASTRIC MUCOSA: A POSSIBLE PRECANCEROUS CHANGE. *(E.)* Sugimura, T.; Kawachi, T.; Kogure, K.; Tokunaga, A.; Tanaka, N.; Sasajima, K.; Koyama, Y.; Hirota, T.; Sano, R. (Natl. Cancer Cent. Res. Inst., Tokyo, Japan). *Proc. Third Int. Symposium Princess Takamatsu Cancer Res. Fund, Japan* 251-261, 1973.

The patterns of four intestine-specific enzymes (sucrase, maltase, trehalase, and lactase) in the sto-

mach mucosa were compared with the histological findings in 134 cases of resected stomach. Samples of stomach mucosa obtained at gastrectomy were treated with solutions of 5% sucrose, maltose, or trehalose and incubated to allow formation of glucose from disaccharides. The mucosal surface was then covered with Tes-Tape, which turned the mucosa green in the presence of disaccharides. Negative reactions for sucrase were obtained in 117 cases of stomach carcinoma, but in 96 cases, the cancer areas were surrounded by tissue giving a positive reaction for sucrase. Two patterns of intestinalization were found: In type A, there were positive reactions for sucrase, maltase, trehalase, and alkaline phosphatase with goblet cells and Paneth cells; and in type B, there were positive reactions for maltase and sucrase with goblet cells, but with negative reactions for trehalase and alkaline phosphatase and no paneth cells. Of the specimens examined, 1/3 showed type A intestinalization, 1/3 showed type B, and 1/3 showed both. In the latter cases, type A intestinalization was generally found in the antral region of the stomach and type B was found in the oral region. Intestinalization might be understood in two ways: it may be an obligatory process in cancerization of the stomach mucosa cells; or the stomach epithelium might be converted to a metastable, unidentified state from which both cancer and intestinalization may arise. At present, it is uncertain which of these possibilities is more likely.

4388 PATHOGENESIS OF GASTRIC LESIONS INDUCED BY ASPIRIN IN THE PYLORUS-LIGATED RAT. *(E.)* Okabe, S.; Takeuchi, K.; Nakamura, K.; Takagi, K. (Fac. Pharm. Sci., Univ. Tokyo, Japan). *Jpn. J. Pharmacol.* 24(3):363-371, 1974.

A standard method for the production of aspirin-induced gastric lesions in rats was elaborated and the mechanisms responsible for the deleterious effects of aspirin were investigated. Male Donryu rats were left unoperated or subjected to laparotomy or pylorus ligation. Immediately after surgery, aspirin (100 mg/kg) and/or 1% carboxymethylcellulose (CMC) solution was administered by gastric intubation. The animals were injected i.v. with pontamine sky blue 6 BX 7 hr later, then sacrificed and the stomachs examined for signs of hemorrhage. In other experiments, sodium bicarbonate, L-glutamine, amylopectine sulfate, sulfated glyptide, or atropine sulfate was administered by gastric intubation after surgery and before aspirin treatment. Compared with unoperated rats, laparotomy significantly reduced the incidence of stomach lesions and pylorus ligation significantly enhanced it. The aspirin-induced lesions in the pylorus ligated animals consisted of severe hemorrhagic erosions of various sizes and nonhemorrhagic mucosal defects. Sodium bicarbonate and L-glutamine strongly inhibited the hemorrhagic effects of aspirin; amylopectine sulfate and sulfated glyptide had less pronounced but significant inhibitory effects; and atropine sulfate had no significant inhibitory effects. Aspirin alone significantly reduced the gastric acidity and had no effect on pepsin activity. Sodium bicarbonate, alone or with aspirin, reduced both the acidity and pepsin activity. L-glutamine, alone or with aspirin, reduced the pepsin activity and increased the gastric acidity. Given with aspirin, amylopectine sulfate and sulfated glyptide reduced the pepsin activity and the gastric acidity. Atropine sulfate had no effect on pepsin activity or gastric acidity. These findings suggest that the effect of pylorus ligation on aspirin-induced gastric lesions is due to the increased acidity induced by the former, that L-glutamine may inhibit the aspirin-induced back diffusion of HCl into the gastric mucosa, and that pepsin plays a minor role in the production of aspirin-induced gastric lesions.

4389 CYTOLOGICAL DIAGNOSIS OF CARCINOMA OF THE STOMACH. *(E.)* Sivanagimani, R.; Reddy, D. B.; Raju, G. C. (Med. Coll., Kurnool, India). *Indian J. Cancer* 11(4):433-436, 1974.

The value of cytodiagnosis in gastric cancer was studied using enzymatic gastric lavage in 78 cases of suspected stomach carcinoma. The fasted patients were given several glasses of water the night and morning before the cytological examination. A ryles tube was passed through the mouth into the stomach and the patient was given a glass of water containing 4 mg trypsin. Gastric lavage was performed and the residual liquid was aspirated, centrifuged, and the residue stained. The results showed 16 positive and two suspected cases of malignancy out of 30 proven cases of cancer. Of the positive cases, two specimens showed signet ring shaped cells indicating an adenocarcinomatous pattern. The other specimens showed large irregular clusters of cells exhibiting nuclear and cytoplasmic abnormalities. The smears of the two cases suggestive of malignancy showed cellular atypism. There was no instance of a false positive diagnosis. The percentage of correct diagnoses obtained in this study is rather low in comparison with the findings reported in the literature.

4390 ACUTE GASTRIC DISEASE AFTER CUTANEOUS THERMAL INJURY. *(E.)* Czaja, A. J.; McAlhany, Jr., J. C.; Andes, W. A.; Pruitt, Jr., B. A. (Brooke Army Med. Cent., Fort Sam Houston, Tex.). *Arch. Surg.* 110(5):600-605, 1975.

To determine the clinical and pathologic characteristics of diffuse erosive gastritis following thermal injury, early and serial endoscopic examinations of the stomach and duodenum were performed on 54 thermally injured patients. A total of 79 examinations were performed without complications using a fiberoptic panendoscope. In addition, various blood values were measured on admission and throughout hospitalization, and gastric biopsy specimens were examined histologically. Diffuse erosive gastritis was discovered as early as 5 hr postinjury in 45 of the patients examined. Acute ulcers were identified in 14 patients and concomitant duodenal disease was present in 34. Microvascular fibrin thrombi were not present, even though five patients had disseminated intravascular coagulation. Of seven patients examined before nasogastric intubation, four with a mean burn size of 59.6% of the total body surface

had diffuse gastritis. Low total serum protein levels were observed in 81% of the patients tested, but were not predictive of mucosal disease. Hemorrhage followed the clinical deterioration of six patients, and in one patient, an ulcer perforated. It is concluded that whereas coagulation abnormalities, nasogastric intubation, and hypoproteinemia may augment mucosal injury, the morphologic and histologic appearance of the lesions suggested a primary ischemic cause resulting from the opening of submucosal shunts or local vasoconstriction.

4391 INFLUENCE OF L-GLUTAMINE ON ASPIRIN-INDUCED GASTRIC LESIONS AND ABSORPTION AS WELL AS ANTIPYRETIC, ANALGESIC AND ANTI-INFLAMMATORY EFFECTS OF ASPIRIN IN RATS AND MICE. *(E.)* Tanaka, H.; Kiyohara, A.; Orima, H.; Suzuki, Y.; Takagi, K.; Okabe, S. (Pharm. Res. Lab., Kyowa Hakko Kogyo Co., Ltd., Shizuoka, Japan). *Jpn. J. Pharmacol.* 24(6): 903-910, 1974.

The effects of L-glutamine on the absorption and pharmacological actions of aspirin and on aspirin-induced gastric lesions were studied in male Wistar rats and male dd mice. Aspirin (100 mg/kg), aspirin plus L-glutamine (1000 mg/kg), or L-glutamine followed by aspirin were given p.o. to pylorus ligated and nonligated rats, some of which had previously been injected s.c. with yeast suspended in physiological saline. In some cases, a phlogistic agent was administered s.c. following aspirin and/or L-glutamine administration and foot pad swelling was measured. In other experiments, aspirin (300 mg/kg) and/or L-glutamine (1000 mg/kg) was administered to mice, which were later injected i.p. with acetic acid or aconitine. L-glutamine markedly suppressed the development of aspirin-induced gastric lesions in the pylorus-ligated rats. In the nonligated rats, aspirin was absorbed rapidly after administration and was maintained in high concentrations in the blood; L-glutamine administration had little effect on the absorption pattern. In the pylorus-ligated rats, however, aspirin administration was followed by a slow increase in the blood salicylate concentration. L-glutamine administration in these animals increased the blood salicylate level. The combined administration of aspirin and L-glutamine had little effect on the antipyretic, analgesic, or anti-inflammatory effects of aspirin in nonligated normal rats and mice. The results indicate that the effect of L-glutamine on aspirin-induced gastric lesions is not due to an inhibition of aspirin absorption and that aspirin retains its pharmacologic potential in the presence of L-glutamine. Therefore, administration of L-glutamine in combination with aspirin is recommended in clinical situations.

4392 FUNCTIONAL STOMACH DISEASE. *(Fr.)* Girard, M. (no affil.). *J. Med. Lyon* 55 (1271):863-869, 1974.

4393 THE STOMACH IN ELDERLY SUBJECTS. *(Fr.)* Roudier, M.; Bodin, F. (no affil.). *Rev. Fr. Gastroenterol.* (99):31-32, 35-36, 38, 1974.

4394 FIBRINOLYTIC ACTIVITY IN THE TISSUE, VASCULAR WALL, AND BLOOD OF PATIENTS WITH GASTRIC DISEASE: PATHOLOGICAL AND PHYSIOLOGICAL FACTORS. *(Jpn.)* Suzuki, T. (Toho Univ. Sch. Med., Japan). *Arch. Jpn. Chir.* 42(4):357-376, 1973.

4395 MASTOCYTOSIS AND URTICARIAL CHANGES IN THE GASTROINTESTINAL TRACT: ENDOSCOPY FINDINGS IN 3 CASES. *(Ger.)* Vetter, D.; Ammann, R.; Deyhle, P.; Schmid, M. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(8):607-608, 1974.

4396 CURRENT CONCEPTS ON PHYSIOLOGICAL CONTROL OF GASTRIC ACID SECRETION. CLINICAL APPLICATIONS. *(E.)* Ivey, K. J. (R. Prince Alfred Hosp., Camperdown, Australia). *Am. J. Med.* 58(3): 389-397, 1975.

4397 PUNCTATE, TRANSENDOSCOPIC, AND PEROPERATIVE pH MEASUREMENTS WITH A RECENTLY DEVELOPED MEASURING DEVICE. *(Ger.)* Miller, G.; Maurer, W.; Trier, R.; Suter, H. R. (Solothurn Munic. Hosp., Switzerland). *Z. Gastroenterol.* 12(8):621-622, 1974.

4398 EXTRAGASTRIC GASTRIN AND GASTRIC HYPERSECRETION. *(Ger.)* Fritsch, W. P.; Hausamen, T. U.; Rick, W.; Kleybrink, H.; Schact, U. (1st Med. Clin., Univ. Dusseldorf, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:521-523, 1974.

4399 EXTRAGASTRIC GASTRIN AND GASTRIC ACID SECRETION. *(Ger.)* Fritsch, W. P.; Hausamen, T. U.; Rick, W.; Kleybrink, H.; Schact, U. (1st Med. Clin. A, Univ. Dusseldorf, Germany). *Med. Welt* 25(31-32):1272-1274, 1974.

4400 EXPERIMENTAL RATIONALE FOR MUCOUS ANTRECTOMY. *(Rus.)* Pantsyrev, Y. M.; Sidorenko, V. I.; Sapelkina, I. M. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Eksp. Khir. Anesteziol.* (5):37-42, 1974.

4401 STUDIES ON TOTAL GASTRECTOMY WITH PRESERVATION OF THE HEPATIC AND THE POSTERIOR CELIAC BRANCHES OF THE VAGUS, ESPECIALLY THE RELATION OF THE HEPATIC BRANCH TO GALLBLADDER FUNCTION AND THE POSTERIOR CELIAC BRANCH TO DUMPING SYNDROME. *(Jap.)* Wakabayashi, N. (Sch. Med., Yamaguchi Univ., Japan). *Arch. Jpn. Chir.* 42(3):211-228, 1973.

4402 NECROTIZING ENTERITIS AFTER ANTRECTOMY AND VAGOTOMY. *(Fr.)* Leger, L.; Chiche, B.; Louvel, A.; Voldman, C. (Cochin Hosp., Paris, France). *J. Chir. (Paris)* 108(4):297-306, 1974.

4403 FUNCTION OF THE CARDIA AND GASTROESOPHAGEAL REFLUX FOLLOWING DISTAL GASTRECTOMY. *(Ger.)* Siewert, R.; Koch, A.; Stuhler, T.; Jennewein, H. M. (Clin. Gen. Surg., Univ. Gottingen, Germany). *Z. Gastroenterol.* 12(8):583-590, 1974.

4404 ORAL ANABOLIC THERAPY FOR GASTRECTOMIZED PATIENTS. *(Ger.)* Bar, W.; Usbeck, W. (Med. Acad., Erfurt, Germany). *Zentralbl. Chir.* 99 (41):1306-1311, 1974.

4405 ARTIFICIAL STOMACH: PATHOPHYSIOLOGY AND SURGICAL POLICY. *(Ger.)* Winkler, R. (Surg. Clin., Univ. Hamburg-Eppendorf, Germany). *Z. Gastroenterol.* 12(6):445-452, 1974.

4406 INVESTIGATION OF Fe KINETICS IN PATIENTS UNDERGOING GASTRECTOMY WITH ESOPHAGODUODENAL ANASTOMOSIS. *(It.)* Dell'Anna, B. (Sch. Semiot. Surg., Univ. Parma, Italy). *Chir. Patol. Sper.* 21(2):173-177, 1975.

4407 COMPARITIVE MULTIFACTORIAL STUDY OF LACTASE ACTIVITY IN THE JEJUNAL MUCOSA OF 40 PATIENTS WHO WERE GASTRECTOMIZED FOR ULCERS BY THE FINSTERER TECHNIQUE AND OF 20 CONTROLS. *(Fr.)* Delmont, J.; Sudaka, P.; Loubiere, M.; Rampal, P. (Cimiez Hosp., Nice, France). *Biol. Gastroenterol. (Paris)* 7(4):301-302, 1974.

4408 CRYOSURGERY AND SUBSEQUENT HEALING IN THE STOMACH AND INTESTINE. *(Ger.)* Breining, H.; Helpap, B.; Langer, S.; Lymberopoulos, S. (Med. Fac., Rhein-Westphal. Tech. Coll., Aachen, Germany). *Z. Gastroenterol.* 12(Suppl.):385-394, 1974.

4409 DECOMPRESSIVE GASTROSTOMY: OUR EXPERIENCE. *(Sp.)* Palma, F.; Luis Palomo, J. (Princess of Spain Hosp. Cent., Jaen, Spain). *Rev. Esp. Enferm. Apar. Dig.* 45(3):244-268, 1975.

4410 CHANGES IN DIGESTION AFTER GASTRECTOMY. *(Rus.)* Diachenko, P. K.; Lysak, Z. A. (Poltava Med. Stomatol. Inst., USSR). *Klin. Khir.* (4):7-12, 1975.

4411 USE OF ANALGESICS IN RADIOLOGY OF THE STOMACH AND DUODENUM. *(Jpn.)* Yamazaki, M.; Inakoshi, H.; Sato, T.; Higuchi, Y.; Chikaraishi, T. (Nigata Univ. Med. Sch., Japan). *Jpn. J. Clin. Radiol.* 49(5):357-370, 1974.

4412 A METHOD TO STUDY CELL PROLIFERATION KINETICS IN HUMAN GASTRIC MUCOSA. *(E.)* Hansen, O. H.; Pedersen, T.; Larsen, J. K. (Bispebjerg Hosp., Copenhagen, Denmark). *Gut* 16(1):23-27, 1975.

4413 LIPID ISLANDS IN THE GASTRIC MUCOSA. *(Ger.)* Kraft, G.; Heilmann, K. (Munic. Hosp., Pforzheim, Germany). *Dtsch. Med. Wochenschr.* 100(3):90-97, 1975.

4414 STUDIES WITH THE HEIDELBERG CAPSULE ON AN IMPROVED GALENIC ANTACID. *(Ger.)* Medee, W.; Muller-Weiland, K. (Natl. Nerve Clin., Salzburg, Germany). *Arzneim. Forsch.* 25(1):122-123, 1975.

4415 ENDOSCOPIC STAINING OF THE STOMACH IN LIVING PATIENTS. *(Ger.)* Tschen, H. H.; Deyhle, P. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(8):622-623, 1974.

4416 DECOMPRESSION PROCEDURES: NASAL INTUBATION OR TEMPORARY GASTROSTOMY. *(Fr.)* Regent, M.; Revil, M.; Delolme, H.; Gounot, J.; Martin, R. (Mutualiste Surg. Clin., St. Etienne, France). *Ann. Anesthesiol. Fr.* 15(Suppl.1):91-98, 1974.

4417 CONSERVATIVE TREATMENT OF STOMACH PHYTOBEZOARS WITH CELLULASE. *(Ger.)* Nauer, E.; Fillippini, L. (Canton. Hosp., Lucerne, Switzerland). *Dtsch. Med. Wochenschr.* 100(6):244-246, 1975.

4418 IMPAIRED GASTRIC DIGESTION. *(Ger.)* Maiwald, L. (Med. Clin., Univ. Wurzburg, Germany). *Z. Gastroenterol.* 13(Suppl.):166-170, 1975.

4419 ACUTE PARTIAL GASTRIC VOLVULUS WHICH REGRESSES SPONTANEOUSLY. *(Ger.)* Winckler, K.; Schuster, R. (Med. Clin., Univ. Gottingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:556-557, 1974.

4420 HEMORRHAGIC GASTROPATHY. (REVIEW OF 11 CASES). *(It.)* Chinaglia, A. (Gen. Reg. Hosp., Treviso, Italy). *Chir. Gastroenterol.* 8(1): 17-27, 1974.

4421 UPPER GASTROINTESTINAL BLEEDING DUE TO GASTRITIS OR UNKNOWN CAUSES. REPORT OF 140 CASES. *(Fr.)* Lataste, J.; Le Picard, P. (Intercommunal Hosp., Creteil, France). *J. Chir. (Paris)* 106(6):541-548, 1973.

4422 GASTRITIS AS A PRECANCEROUS CONDITION. *(Rus.)* Khonelidze, G. B.; Iakovleva, I. A. (Moldavian Res. Inst. Oncol., Kishinev, USSR). *Vopr. Onkol.* 20(12):34-38, 1974.

4423 PHLEGMONOUS GASTRITIS. *(Ger.)* Bron, B. A.; Pelloni, S.; Krejs, G.; Deyhle, P.; Blum, A. L. (Triemli Munic. Hosp., Zurich, Switzerland). *Z. Gastroenterol.* 12(8):605-606, 1974.

4424 DRUGS AND GASTRIC BLEEDING: RESULTS OF A ONE-YEAR PROSPECTIVE STUDY. *(Ger.)* Kapp, F.; Baerlocher, C.; Fahrlander, H. (Med. Dep., Univ. Basel, Switzerland). *Z. Gastroenterol.* 12(8):602-603, 1974.

4425 EROSIONS AS THE SOURCE OF ACUTE UPPER GASTROINTESTINAL BLEEDING. *(Ger.)* Lammli, J.; Funk, C.; Knoblauch, M. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(8):601, 1974.

4426 EROSIVE GASTRITIS AND EROSIVE DUODENITIS. *(Ger.)* Stocker, H.; Baumgartner, R.; Gassmann, R.; Hefti, M. L.; Leuthold, E. (Zurich Endosc. Coll., Switzerland). *Z. Gastroenterol.* 12(8):600-601, 1974.

4427 THE THERAPEUTIC EFFECT AND EFFECT ON GASTROINTESTINAL FUNCTION OF METHYLMETHIONINE SULFONIUM CHLORIDE (VITAMIN U) IN THE TREATMENT OF PATIENTS WITH CHRONIC GASTRITIS. *(Rus.)* Trusov, V. V.; Oreshkov, T. M. (Izhevsk Med. Inst., USSR). *Sov. Med.* (8):121-124, 1974.

4428 SERUM LACTATE DEHYDROGENASE ISOENZYMES IN PATIENTS WITH CHRONIC GASTRITIS ASSOCIATED WITH HYPOCHLORHYDRIA OR STOMACH CANCER. *(Rus.)* Lomachenkov, V. D. (Smolensk Med. Inst., USSR). *Ter. Arkh.* 46(8):126-130, 1974.

4429 IMMUNOGLOBULIN CONTENT OF GASTRIC JUICE FROM PATIENTS WITH CHRONIC ATROPHIC GASTRITIS AND CIRRHOSIS. *(Ger.)* Schmidt-Wilcke, H. A.; Lapp, M.; Lorenz-Meyer, H. (Med. Clin., Univ. Marburg/Lahn, Germany). *Z. Gastroenterol.* 12(6):459-466, 1974.

4430 THE USE OF THIOAMINES IN THE FOLLOW-UP OF PATIENTS WITH GASTROINTESTINAL NEOPLASMS AND CHRONIC GASTRITIS. *(Rus.)* Lorman, T. B. (Reg. Oncol. Dyspens., Pskov, USSR). *Lab. Delo* (3):126-164, 1975.

4431 GASTROCOLIC FISTULA DUE TO BENIGN GASTRIC ULCER. A REPORT OF TWO CASES. *(E.)* Harned, R. K.; Pergam, C. J. (Univ. Nebraska Med. Cent., Omaha). *Am. J. Gastroenterol.* 63(2):158-161, 1975.

4432 COMPARITIVE MORPHOMETRIC ANALYSIS OF CHANGES IN THE GLANDULAR APPARATUS OF THE PYLOROANTRAL PART OF THE STOMACH IN PEPTIC ULCER AND CANCER. *(Rus.)* Samsonov, V. A.; Koroleva, N. P.; Sharshavitsky, G. A. (I. P. Pavlov 1st Leningrad Med. Inst., USSR). *Arkh. Patol.* 36(9):31-35, 1974.

4433 CHRONIC GASTRIC EROSIONS: FOLLOW-UP STUDY. *(Ger.)* Aenishanslin, H. W.; Stadler, G. A. (Dep. Intern. Med., Univ. Basel, Switzerland). *Z. Gastroenterol.* 12(8):598, 1974.

4434 CLASSIFICATION OF GASTRIC EROSIONS. *(Ger.)* Rosch, W. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Z. Gastroenterol.* 12(8):597-598, 1974.

4435 ENDOSCOPIC FOLLOW-UP OF EROSIONS IN THE GASTRIC MUCOSA. *(Ger.)* Krentz, K.; Gohrband, G. (Luisen Hosp., Aachen, Germany). *Z. Gastroenterol.* 12(8):599-600, 1974.

4436 ACUTE POSTTRAUMATIC GASTRIC DISEASE. I. THE VIEWPOINT OF THE RESUSCITATING PHYSICIAN. *(Fr.)* du Cailar, J. (St. Eloi Clin., Montpelier, France). *Ann. Anesthesiol. Fr.* 15(Suppl. 1):23-27, 1974.

4437 SALICYCLIC ACID AND OCCULT HEMMORRHAGES IN THE GASTROINTESTINAL TRACT. *(Ger.)* Engelhardt, A. (no affil.). *Arzneim. Forsch.* 24(10):1594-1600, 1974.

4438 THE INDUCTION OF GASTROINTESTINAL HEMORRHAGES IN THE RAT BY ACETYLSALICYLIC ACID (ASA) AND AN ASA-CONTAINING COMBINATION. *(Ger.)* Kriegel, H.; Schramm, E. (Inst. Radiol. Environ. Res., Munich, Germany). *Arzneim. Forsch.* 24(10):1590-1594, 1974.

4439 STRESS ULCER IN RATS WITH PORTOCAVAL ANASTOMOSIS (PCA). *(Ger.)* Schumpelick, V.; Rauchenberger, B. (Surg. Clin., Univ. Hamburg, Germany). *Langenbecks Arch. Chir.* 338(1):1-8, 1975.

4440 MALIGNANT GASTRIC LEIOMYOBLASTOMA. *(E.)* Uhrich, G. I. (La Crosse Clin., Wis.). *Abdom. Surg.* 17(3):73-76, 1975.

4441 THE EFFECTS OF FAST NEUTRONS ON INOPERABLE CARCINOMA OF THE STOMACH. *(E.)* Catterall, M.; Kingsley, D.; Lawrence, G.; Grainger, J.; Spencer, J. (Med. Res. Counc. Cyclotron Unit, London, England). *Gut* 16(2):150-156, 1975.

4442 STOMACH CANCER AND ECOLOGIC FACTORS IN JAPAN. *(E.)* Takahashi, E. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 113(2):129-133, 1974.

4443 α_1-ACID GLYCOPROTEIN IN GASTRIC CANCER JUICE. *(E.)* Rapp, W.; Heim, M.; von Mikulicz-Radecki, J. G.; Ludwig, R. (Med. Clin., Univ. Heidelberg, Germany). *Klin. Wochenschr.* 53(3):139-141, 1975.

4444 NODULAR LYMPHOID HYPERPLASIA OF THE STOMACH AND SMALL INTESTINE WITH HYPOGAMMAGLOBULINAEMIA. *(E.)* Munro, A.; Simpson, J. G. (Dep. Surg. Pathol., Univ. Aberdeen, Scotland). *Br. J. Surg.* 61(12):953-954, 1974.

4445 DIAGNOSIS AND TREATMENT OF BENING NONEPITHELIAL GASTRIC TUMORS. *(Rus.)* Chernousov, A. F.; Gukasian, A. A.; Stranadko, E. F.; Yevtikhiyeva, E. P.; Mlynchik, V. E.; Shengelaia, T. F.; Penkov, L. K. (All-Union Res. Inst. Clin. Exp. Surg., Moscow, USSR). *Khirurgiia (Mosk.)* (1): 60-64, 1974.

4446 MORPHOGENETIC CHARACTERISTICS OF EARLY CANCER OF THE STOMACH. *(Rus.)* Nesvetov, A. M.; Rokhlin, E. A.; Shusterov, M. I. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Arkh. Patol.* 36(9): 24-31, 1974.

4447 DIAGNOSIS OF PRIMARY MULTIPLE CANCER OF THE ESOPHAGUS AND STOMACH. *(Rus.)* Topuzov, E. G.; Uchvatkin, V. G. (Altai Med. Inst., Barnaul, USSR). *Vestn. Khir.* 113(10):19-23, 1974.

4448 GASTROSCOPIC DIAGNOSIS OF STOMACH TUMORS UNDER OUTPATIENT CONDITIONS. *(Rus.)* Babichev, S. I.; Briskin, B. S.; Komarovsky, V. S.; Fomin, V. B. (I. I. Dzhanelidze Res. Inst. Emerg. Med., Leningrad, USSR). *Vestn. Khir.* 113(9):3-7, 1974.

4449 LYMPHOSARCOMA OF THE STOMACH: DIAGNOSTIC VALUE OF GASTROSCOPY AND GASTROBIOPSY WITH DIRECT VISUAL MONITORING. *(Fr.)* Cornet, A.; Barbier, J. P.; Carnot, F. (Laennec Hosp., Paris, France). *Sem. Hop. Paris* 50(25):1673-1682, 1974.

4450 TRANSFORMATION OF THE GASTRIC AND INTESTINAL EPITHELIUM IN MAN AND EXPERIMENTAL ANIMALS. *(Rus.)* Zufarov, K. A.; Baibekova, E. M. (Tashkent Med. Inst., USSR). *Arkh. Anat. Gistol. Embriol.* 67(10):49-56, 1974.

4451 THE DIAGNOSIS OF AND THE PROGNOSIS IN SCIRRHOUS CARCINOMA OF THE STOMACH. *(Ger.)* Rosch, W.; Herzig, M.; Schwemmle, K. (Med. Clin., Univ. Erlange,-Nuremberg, Erlangen, Germany). *Bruns Beitr. Klin. Chir.* 211(6):423-230, 1974.

4452 NONEPITHELIAL GASTRIC NEOPLASMS: STUDY OF 22 ORIGINAL CASES. *(Itl.)* Balestrazzi, A.; Balestrazzi, N. (Hosp. Consortium, Bari, Italy). *Chir. Gastroenterol.* 8(2):159-186, 1974.

4453 AGE OF PATIENTS WHO DIED OF CARCINOMA OF THE STOMACH AND LOCALIZATION OF THE TUMOR. *(Pol.)* Duda, K.; Kowalczyk, J. (Dep. Surg. II, Med. Acad., Kracow, Poland). *Pol. Przegl. Chir.* 46(11): 1569-1574, 1974.

4454 PRIMARY GASTRIC SARCOMA. *(Ger.)* Schumpelick, V. (Surg. Clin., Univ. Hamburg, Germany). *Z. Gastroenterol.* 12(7):519-528, 1974.

4455 PSEUDOXANTHOMA (CHOLESTEATOMA) OF THE STOMACH. *(Ger.)* Rosch, W. (Med. Clin., Univ. Erlangen, Germany). Z. *Gastroenterol.* 12(8): 606-607, 1974.

4456 SECOND GASTRECTOMY FOR SARCOMYOMA OCCURRING 28 YEARS AFTER THE FIRST OPERATION. *(Pol.)* Szpakowski, J. (Acad. Med., Krakow, Poland). *Przegl. Lek.* 31(7):712-713, 1974.

4457 HISTOLOGY AND HISTOGENESIS OF EARLY CANCER OF THE STOMACH. *(Ger.)* Grundmann, E. (Pathol. Inst., Univ. Munster/Westphalia, Germany). *Therapiewoche* 24(36):3824, 3827-3828, 3831, 1974.

4458 EARLY MULTICENTRIC CARCINOMA OF THE STOMACH: 2 CASES. *(Ger.)* Gassman, R.; Leuthold, E.; Siebermann, R.; Pfenninger, E. (Triemli Munic. Hosp., Zurich, Switzerland). Z. *Gastroenterol.* 12(8):604-605, 1974.

4459 CASE REPORT OF AN EARLY GASTRIC CARCINOMA DEVELOPED IN THE REMNANT STOMACH. *(Jpn.)* Yamagishi, T.; Takamura, U.; Kasaoka, C.; Kono, T.; Kaifu, S.; Morioka, M.; Suzuki, O.; Ito, S.; Hayano, I. (Fujisawa City Hosp., Kanagawa Prefect, Japan). *Jpn. J. Can. Clin.* 20(6):499-503, 1974.

4460 CLINICO-EPIDEMIOLOGICAL STUDY OF STOMACH CANCER: SEX AND AGE DIFFERENCES. *(Jpn.)* Kurita, H. (Sch. Med., Nagoya Munic. Univ., Japan). *Jpn. J. Cancer Clin.* 20(8):580-593, 1974.

4461 DEFIBRINATION SYNDROME IN GASTRIC TUMORS. *(Ger.)* Misz, M.; Szabo, A.; Batonyi, E.; Tompa, G.; Papp, L. (Med. Univ., Debrecen, Hungary). *Munch. Med. Wochenschr.* 117(8):287-290, 1975.

4462 RESULTS OF OPERATIVE TREATMENT OF STOMACH CARCINOMA. *(Pol.)* Adamus, J.; Przywara, S.; Pysz, H.; Gebel, B.; Plewa, T. (Inst. Oncol., Gliwice, Poland). *Pol. Przegl. Chir.* 46(11):1531-1536, 1974.

4463 THE PROBLEM INVOLVED IN DELIMITING GASTRIC GRANULOBLASTOMA. *(Ger.)* Feichter, G. E.; Gnandiger, H. P. (Pathol. Inst., Johannes Gutenberg-Univ., Mainz, Germany). *Bruns Beitr. Klin. Chir.* 221(7):525-530, 1974.

4464 PRIMARY GASTRIC SARCOMA. *(Ger.)* Schumpelick, V. (Surg. Clin., Univ. Hamburg, Germany). *Bruns Beitr. Klin. Chir.* 221(6):431-444, 1974.

4465 CLINICOPATHOLOGICAL STUDY ON CANCER OF THE CARDIA. *(Jpn.)* Seki, H. (Gunma Univ. Sch. of Medicine, Maebashi, Japan). *Kitakanto Igaku (Kitakanto Medical J.)* 24(4):189-206, 1974.

4466 EARLY GASTRIC CANCER. *(Hun.)* Oshima, H. (Clin. Surg., Free Univ. Berlin, Steglitz, Germany). *Magy. Onkol.* 18(1):13-18, 1974.

4467 RELIABILITY OF STATISTICAL DATA ON THE INCIDENCE OF GASTRIC CARCINOMA. *(Hun.)* Schaefer, K. (Clin. Intern. Med., Justus Liebig Univ., Giessen, Germany). *Magy. Onkol.* 18(1):2-8, 1974.

4468 GASTRIC LYMPHOSARCOMA. *(Fr.)* Dreze, C.; Lombard, R. (St. Joseph Clin., Liege, Belgium). *Rev. Med. Liege* 29(22):688-691, 1974.

4469 CONGENITAL HYPERTROPHIC PYLORIC STENOSIS 9 YEAR CLINICAL STUDY OF 42 CASES). *(E.)* Joseph, T. P.; Nair, R. R. (S.A.T. Hosp., Trivandrum, India). *Indian J. Surg.* 36(6):221-223, 1974.

4470 CURRENT ASPECTS OF HYPERTROPHIC PYLORIC STENOSIS IN INFANTS. *(Fr.)* Juskiewenski, S.; Ghisolfi, J.; Vayasse, P.; Bacque, P.; Veyrie, J. (no affil.). *Rev. Med. Toulouse* 10(6):1139-1148, 1974.

4471 PHYSIOLOGY AND PATHOPHYSIOLOGY OF GASTRIN. *(E.)* Grossman, M. I. (no affil.). *Clin. Gastroenterol.* 3(3):533-538, 1974.

4472 GASTRIC CARCINOIDS SIMULATING BENIGN POLYPS. TWO CASES DIAGNOSED BY ENDOSCOPIC BIOPSY. *(E.)* Gueller, R.; Haddad, J. K. (VA Hosp., San Francisco, Calif.). *Gastrointest. Endosc.* 21(4): 153-155, 1975.

4473 INVOLVEMENT OF THE STOMACH WITH WALDENSTROM'S MACROGLOBULINEMIA. *(E.)* Cline, W. S.; Imbeau, S. A.; Morrissey, J. F. (VA Hosp., Madison, Wis.). *Gastrointest. Endosc.* 21(4):166-167, 1975.

4474 XANTHOMAS OF THE STOMACH. *(E.)* McCaffery, Jr., T. D. (Louisiana State Univ. Med. Cent., New Orleans). *Gastrointest. Endosc.* 21(4): 167-168, 1975.

4475 INCARCERATED JEJUNOGASTRIC RETROGRADE INTUSSUSCEPTION ASSOCIATED WITH HEMATEMESIS: GASTROSCOPIC OBSERVATIONS. *(E.)* Dagradi, A. E.; Alaama, A.; Ruiz, R. (VA Hosp., Long Beach, Calif.). *Gastrointest. Endosc.* 21(4):168-179, 1975.

4476 GASTRIC SARCOIDOSIS PRESENTING AS ANTRAL SCARRING. *(E.)* Blackstone, M. O.; Dhar, G. J.; Mizuno, H.; Para, M. F. (Dep. Med., Univ. Chicago, Ill.). *Gastrointest. Endosc.* 21(4):184, 1975.

4477 PYLORIC CHANNEL DUPLICATION CAUSING GASTRIC OUTLET OBSTRUCTION. *(E.)* Bertram, P. D. (Cookeville Gen. Hosp., Tenn.). *Gastrointest. Endosc.* 21(4):183, 1975.

4478 RECOVERY FROM CURLING'S ULCER IN CHILDHOOD AFTER SURGERY. *(Heb.)* Motovic, A.; Man, B.; Kraus, L.; Sadan, N. (Meir Hosp., Kfar Saba, Israel). *J. Isr. Med. Assoc.* 87(11):496-497, 530, 1974.

4479 ALKALINE REFLUX GASTRITIS AS A CAUSE OF DISTRESS AFTER GASTRIC OPERATIONS. *(E.)* Landor, J. H.; Meckeler, K. J. H. (Rutgers Med. Sch., Piscataway, N.J.). *J. Med. Soc. NJ* 72(6):487-489, 1975.

4480 SIMULTANEOUS OCCURRENCE OF CONGENITAL PYLORIC WEB AND HIATAL INSUFFICIENCY IN AN INFANT. *(E.)* Kekomaki, M.; Kuitunen, P. (Child. Hosp., Helsinki, Finland). *Helv. Paediatr. Acta* 29(6):595-598, 1974.

4481 PREVALENCE OF ATROPHIC FUNDAL AND ANTRAL GASTRITIS IN RANDOMLY SELECTED ESTONIANS (PRELIMINARY REPORT). *(E.)* Villako, K.; Tamm, A.; Savisaar, E.; Ruttas, M. (Tartu State Univ., Estonia, USSR). *Acta Hepatogastroenterol. (Stuttg.)* 22(1): 62-63, 1975.

See also, 3798, 3866, 4192, 4215, 4216, 4220, 4221, 4222, 4223, 4224, 4225, 4284, 4290, 4293, 4302, 4305, 4365, 4518, 4551, 4573, 4889, 5043, 5094, 5099, 5103, 5126, 5168, 5185.

STOMACH

Peptic Ulcers

4482 SERUM PYRIDOXAL IN ACTIVE PEPTIC ULCERATION. *(E.)* Sanderson, C. R.; Davis, R. E. (R. Perth Hosp., Australia). *Gut* 16(3):177-180, 1975.

Fasting serum pyridoxal was assayed by an automated microbiological system in 50 patients (24 men and 26 women, mean age 53 yr) with endoscopically confirmed peptic ulceration. Serum pyridoxal was below normal in 28 of 30 patients with gastric ulceration, one of 14 with duodenal ulceration, one of four with pyloric canal ulceration, and each of two patients with both gastric and duodenal ulceration. Mean serum pyridoxal levels in gastric ulcer and duodenal ulcer patients were 14.7 and 30.8 nM/liter,

resp. Although the gastric acid group had a lower mean serum folate level compared with the duodenal ulcer group, no correlation between individual levels was found. The low serum pyridoxal level in patients with gastric ulceration and the normal level in those with duodenal ulceration could not be explained by differences in age, sex, drug use, alcohol intake, or diet. The differences may reflect differing biochemical bases between the two groups, or it may be the result of the ulceration itself.

4483 EFFECT OF METIAMIDE ON THE RESPONSE TO SECRETIN AND CHOLECYSTOKININ IN MAN. *(E.)* Thjodleifsson, B.; Wormsley, K. G. (Dep. Ther., Univ. Dundee, Scotland). *Gut* 16(1):33-35, 1975.

The effects of i.v. metiamide on the pancreatic exocrine response to i.v. infusion of secretin (1 clinical U/kg/hr) plus cholecystokinin (CCK, 1 Ivy U/kg/hr) was studied in eight patients with duodenal ulceration. Five patients received a continuous infusion of 200 mg/hr metiamide for 1 hr; the remaining three were given a loading dose of 75 mg metiamide prior to continuous infusion of 100 mg/hr metiamide and hormones. The results of the two metiamide schedules did not differ significantly. Metiamide did not alter the secretion of bicarbonate and water. Trypsin secretion in response to secretin plus CCK decreased significantly only during the second 30 min of the 1-hr test. Metiamide virtually abolished gastric acid response to the hormones but had no consistent effect on the cholecystokinetic response. The cause of marked difference between metiamide effects on gastric and pancreatic secretion is not clear. Minimal pancreatic inhibitory effects would be expected if the secretory response to exogenous stimulants depends on interaction between stimulant and receptors and if metiamide acts by competing with gastrin (or hormone) receptors. Alternatively, no inhibitory effect of metiamide on pancreatic secretion would be expected if histamine mediates gastric secretory stimulation by exogenous stimulants but is not involved in mediating the effect of secretin and CCK on the pancreas.

4484 DUODENOGASTRIC REFLUX: A CAUSE OF GASTRIC MUCOSAL HYPERAEMIA AND SYMPTOMS AFTER OPERATIONS FOR PEPTIC ULCERATION. *(E.)* Keighley, M. R. B.; Asquith, P.; Alexander-Williams, J. (Gen. Hosp., Birmingham, England). *Gut* 16(1):28-32, 1975.

The association between duodenogastric reflux, gastritis, and symptoms was investigated in 30 patients with or without dyspepsia 1-15 yr after gastric ulcer surgery. Patients with biliary disease, hiatus hernia, or recurrent ulceration were excluded from the study. The 30 patients (25 men and five women, mean age 42 yr) were assessed by a symptomatic score, measurement of bilirubin and sodium concentrations in fasting gastric juice, gastric biopsy, and the presence of radiological reflux. In 15 patients with a symptom score of less than median, gastric bilirubin levels were less than 1 mg/ml in 80%; severe endoscopic changes were seen in only one patient and reflux was not observed. Of the 15 patients with a symptom score in excess of the median, 12 had a bilirubin concentration greater than 1 mg/100 ml and four of these had a level in excess of 4 mg/100 ml. Radiological reflux was present in 60% of this group and severe mucosal hyperemia in 50%. There was a significant correlation between symptoms, gastric hyperemia, and duodenal reflux ($P<0.02$). Symptoms closely correlated with duodenal reflux included bile vomiting, nausea, epigastric pain after meals, bile regurgitation, heartburn, and fatty intolerance. Further gastric surgery other than a biliary diversion is likely to aggrevate rather than improve the symptoms.

4485 THE NATURAL HISTORY OF GASTRIC ULCER IN A COMMUNITY: A FOUR-YEAR STUDY. *(E.)* Mowat, N. A. G.; Needham, C. D.; Brunt, P. W. (Dep. Med., Univ. Aberdeen, Scotland). *Q. J. Med.* 44(173): 45-56, 1975.

All (151) adult patients in northeast Scotland with supposedly benign gastric ulcer first diagnosed in 1967 were studied after a minimum of 4 yr, with a 98% follow-up. The incidence and male:female ratio both increased with advancing age. Ulcers of the body of the stomach were twice as common as ulcers of the antral region. Major coincidental disease (largely cardiovascular, respiratory, or locomotor) was present in 59% of the patients and was the principal cause of 25 of the 29 deaths in the series. Twenty-one patients presented with bleeding but only one other bled subsequently; one patient presented with perforation of gastric ulcer; and two ulcers proved to be malignant. Neither presentation nor course was significantly affected by associated duodenal ulceration (33% of cases). There was a direct correlation between short duration of symptoms and increasing age and mortality. Forty-four (55%) of 82 medically treated patients and 41 (60%) of 69 surgically treated patients were alive and symptom-free at the end of the follow-up period; the respective mortalities were 18.2% and 20.3%. For the whole series, life expectancy was significantly reduced in females (15 deaths *versus* 6.03 expected), but not in males.

4486 CHOLINERGIC STIMULATION OF THE LOWER ESOPHAGEAL SPHINCTER IN PATIENTS WITH VAGOTOMY AND ANTRECTOMY. *(E.)* Higgs, R. H.; Castell, D. O. (US Nav. Hosp., Philadelphia, Pa.). *Am. J. Dig. Dis.* 20(3):195-200, 1975.

The effect of bethanechol on lower esophageal sphincter (LES) pressure was determined in 10 vagotomized and antrectomized patients (eight men and two women, mean age 45 yr). Both s.c. (0.08 mg/kg) and p.o. (25 mg) administration of bethanechol caused a significant increase in LES pressure in these patients. Sphincter pressure in patients who received the drug s.c. increased from a mean basal level of 16.5 to 41.2 mm Hg at 25 min. Hypotensive basal LES pressure in four patients rose from 8.0 to 41.0 mm Hg. In the six patients given bethanechol p.o., sphincter pressure increased from 11.2 to 19.1 mm

Hg at 75 min. Three of these patients had hypotensive sphincter pressure plus symptomatic gastroesophageal reflux and endoscopic evidence of esophagitis. Following p.o. bethanechol, sphincter pressure rose from 8.0 to 18.3 mm Hg. P.o. administered bethanechol produced a sustained increase in LES pressure throughout a 90-min study period. These studies suggest that cholinergic stimulation of the LES with bethanechol may be of therapeutic benefit in vagotomized and antrectomized patients with gastroesophageal reflux.

4487 DISPERSION ANALYSIS OF PROGNOSTIC AND RELAPSING FACTORS OF DUODENAL ULCER BY USE OF ELECTRON COMPUTER. *(E.)* Kawai, K.; Kohli, Y. (Kyoto Prefect. Univ. Med., Japan). *Am. J. Gastroenterol.* 63(1):20-28, 1975.

Complete healing of duodenal ulcers and ulcer scars was documented endoscopically in 109 of 192 patients (92.2% male) studied over a 3-yr period. The 192 cases included 91 with a single duodenal ulcer, 57 with kissing ulcers, and three with multiple ulcers. The shape of single ulcers was round in 49 cases, irregular in 24, long linear in 10, and short linear in eight. After healing, ulcers recurred in 25 cases. Computer analysis of morphologic data showed that linear ulcers were more intractable to healing than round or irregularly shaped ulcers. Increased age was associated with an increased incidence of linear ulcers and a decreased incidence of round ulcers. Ulcers with a marginal swelling appeared to heal more readily than those without it. Number, distribution of lesions, and degree of radiological deformity of the duodenal cap were of no prognostic value. However, kissing ulcers and scars with redness or convergency of mucosal folds showed a tendency to recur. Endoscopy was more useful than radiological examination in description of location, number, shape, and/or stage of duodenal ulcers.

4488 pH DEPENDENCE OF ACID SECRETION AND GASTRIN RELEASE IN NORMAL AND ULCER SUBJECTS. *(E.)* Walsh, J. H.; Richardson, C. T.; Fordtran, J. S. (Dep. Med., Univ. California, Los Angeles). *J. Clin. Invest.* 55:462-468, 1975.

Studies were performed on normal subjects and duodenal ulcer patients to assess the effect of gastric acid concentration on the rate of gastric acid secretion and endogenous gastrin release in response to an amino acid or a steak meal. The rate of gastric acid secretion was estimated by measuring the rate of sodium bicarbonate infusion needed to keep intragastric pH constant. Preliminary studies on five normal subjects showed that the secretion rate with an intragastrically instilled amino acid-cornstarch meal was highest at pH 5.5 and was 60% of that produced by a steak meal at the same pH. Progressive lowering of intragastric pH was associated with a stepwise reduction in acid secretion, with secretion at pH 2.5 only half as great as at pH 5.5. The amino acid meal produced increases in serum gastrin that were also less marked than those produced by a steak meal. Increase in serum gastrin over basal concentrations was similar at pH 5.5, 4.0, and 3.0; a measurable increase was not obtained when the meal was maintained at pH 2.5. Six patients with duodenal ulcers (three men and three women, mean age 46.5 yr) were compared with seven normal subjects (three women and four men, mean aged 31.4 yr) at pH 5.5 and 2.5. At both pH values, ulcer patients secreted more acid throughout the 2-hr period following instillation of amino acid meal than did the normal subjects. More important, the degree of inhibition at pH 2.5 was significantly less in ulcer patients. During the second postprandial hr at pH 2.5, acid secretion was suppressed 70% in the controls and only 30% in the patients. These findings suggest a defect in autoregulation of gastrin release and gastric acid secretion at low pH in ulcer patients which may play a role in the pathogenesis of this disease.

4489 ULTRA-STRUCTURAL CHANGES IN THE SMOOTH MUSCULATURE OF ANTRUM AND PYLORUS IN PRESENCE OF ULCUS VENTRICULI. *(E.)* van der Zypen, E. (Inst. Anat., Univ. Basel, Switzerland). *Res. Exp. Med. (Berl.)* 164(1):1-17, 1974.

The smooth musculature of the antrum and pylorus of seven patients with one or more stomach ulcers was examined by transmission electron microscopy. The corresponding regions from five normal organ donors served as control material. Electron microscopically visualizable changes were found at distances of up to 180 mm from the site of ulcerous lesions on the smooth muscle cells of the inner and outer longitudinal musculature, and on blood vessels. In the majority of cases, condensation of sarcoplasmic ground substance and myofilaments was observed in combination with distention of hypolemmal vesicles and proliferation of dense bodies. These changes are interpreted as an expression of a pronounced isometric contraction of smooth musculature in the presence of ulcera ventriculi: following glutaraldehyde fixation, the normal stomachs were invariably in a state of relaxation, whereas stomachs with ulceration were partially in a state of isometric contraction. Other changes observed in the presence of stomach ulcer were swelling of the endoplasmic reticulum of smooth muscle cells, lysis of myofilaments, and lacunae. Vacuolization of the endoplasmic cisternae caused displacement of other cell organelles and of the myofilaments. The swelling of the endoplasmic spaces and lysis of the myofilaments indicate degeneration of the muscle cells. The capillary endothelial cells showed degenerative changes that lead to arrest of cytopemtpic processes. In the arteriolar regions, a strong contraction of the smooth musculature frequently resulted in complete occlusion of the vascular lumen. Though further systematic studies are needed, these findings substantiate light microscopy observations on the antral and pyloric musculature in the presence of stomach ulcer.

4490 FAILURE OF SECRETIN RELEASE IN PATIENTS WITH DUODENAL ULCER. *(E.)* Bloom, S. R.; Ward, A. S. (Middlesex Hosp., London, England). *Br. Med. J.* 1(5950):126-127, 1975.

Secretin release was studied in 23 patients with duodenal ulcer (21 men and 2 women, mean age 42 yr) and 12 normal subjects (10 men and 2 women, mean age 38 yr). I.v. infusion of pentagastrin (6 μg/kg/hr) produced a plateau of gastric acid secretion that was significantly higher in the duodenal ulcer patients than in the normal subjects. Intraduodenal infusion of 40 ml of 0.1 M HCl resulted in a similar absolute fall in acid output in the two groups, but the rise in plasma secretin levels after intraduodenal acid infusion was significantly higher in controls than in duodenal ulcer patients (52.6 ng/liter *versus* 37.5 ng/liter at 6 min). Patients with symptoms of duodenal ulcer for less than 10 yr and those with a history over 10 yr had a very similar 10-min secretin release (274 and 273 ng/liter/min, resp.). The nine duodenal ulcer patients who smoked 20 or more cigarettes a day had a particularly reduced secretin release compared with that of patients who smoked less than 15 cigarettes a day (216 *versus* 312 ng/liter/min). No correlation was apparent between the amount of secretin released by intraduodenal acid and either the degree of gastric inhibition or the level of the initial gastric acid plateau. These results demonstrate that patients with a duodenal ulcer have an impaired secretin release in spite of requiring greater pancreatic bicarbonate output to neutralize a larger gastric acid load.

4491 THE EFFECT OF AN H_2-RECEPTOR ANTAGONIST ON FOOD-STIMULATED ACID SECRETION, SERUM GASTRIN, AND GASTRIC EMPTYING IN PATIENTS WITH DUODENAL ULCERS: COMPARISON WITH AN ANTICHOLINERGIC DRUG. *(E.)* Richardson, C. T.; Bailey, B. A.; Walsh, J. H.; Fordtran, J. S. (Univ. Texas Health Sci. Cent., Dallas). *J. Clin. Invest.* 55:536-542, 1975.

The effects of isopropamide (an anticholinergic drug) and metiamide (an H_2-receptor antagonist) on food-stimulated acid secretion were compared. Seven duodenal ulcer patients (two women and five men, mean age 41.7 yr) were stimulated by a steak meal, and acid secretion was measured by *in vivo* intragastric titration. A maximum tolerated p.o. dose of isopropamide (20-40 mg/dose) inhibited food-stimulated acid secretion by a maximum of 35%. In contrast, a 400-mg dose of p.o. metiamide produced no side effects and inhibited secretion by 96%. A maximum tolerated dose of isopropamide and 300 mg metiamide suppressed food-stimulated acid secretion to a greater extent than 400 mg metiamide alone (88 mEq reduction *versus* 63 mEq). Neither isopropamide nor metiamide affected serum gastrin concentration or gastric emptying. If given before meals, metiamide was most effective during the early posprandial period, whereas if given after meals, it was most effective during the second hr after eating. The duration of action was at least 3 hr. These results indicate that histamine has a dominant role in mediating food-stimulated acid secretion and that the specific inhibitory effect of metiamide on acid secretion is not mediated through inhibition of gastrin release.

4492 THE EFFECT OF HISTAMINE ON GASTRIC SECRETION IN PATIENTS WITH PEPTIC ULCER. *(Rus.)* Voinova, M. Kh.; Kliminskii, I. V. (2nd Moscow Med Inst., USSR). *Khirurgiia (Mosk.)* (4):58-62, 1974.

Studies were carried out on gastric acid secretion and the histamine content of the gastric juice, blood and tissues of 24 males with peptic ulcer (18 had ulcers in the duodenum, 6 in the stomach). Six healthy adults served as the controls. Tests were made to determine the basal acid output (BAO) and maximal acid output (MAO) after stimulation with exogenous histamine (0.04 μg/kg). In patients with duodenal ulcer, the histamine content in the blood did not differ significantly from that of the controls, while excessive acid secretion was observed in the stomach: BAO 6.25±0.81 mEq/hr and MAO 37.22±0.64 mEq/hr (in healthy adults: 2.1±0.64 and 10.5±3.0 mEq/hr, resp.). Basal histamine secretion with the gastric juice averaged 132.8±27.8 μg/hr and in stimulated secretion, 160.23±6.4 μg/hr. Drug-induced (benzohexonium and atropine) and surgical vagotomy significantly decreased histamine secretion and the histamine content in the gastric juice. A correlation was found between acid and histamine content in the gastric juice in patients with stomach ulcer. It is suggested that histamine content in the gastric juice in patients with duodenal ulcer is closely related to the tonus of the vagus nerve.

4493 CERTAIN PROBLEMS OF HEREDITY ASSOCIATED WITH PEPTIC ULCER. *(Rus.)* Frolkis, A. V.; Somova, E. P. (Petrozavodsk University, USSR). *Klin. Med. (Mosk.)* 52(8):120-124, 1974.

Family studies of 124 peptic ulcer patients showed that in 35 (28.2%) families relatives of the propositi had a prior history of the disease. Adverse family histories (peptic ulcer, gastritis, epigastric pain, intestinal cancer) among the patients' families was 5.5 times greater than among the families of 124 controls with no gastrointestinal disease (52.4% and 9.6%, resp.). There were significantly more peptic ulcer patients with the O(I) blood group and among the nonsecretors of A, B and AB antigens. Starch gel electrophoretic studies of serum alkaline phosphatase isoenzymes from peptic ulcer patients showed that the slow-moving B component was absent in 58% of the patients, mostly those with the A(II) blood group. Serum pseudocholinesterase in peptic ulcer patients was significantly lower than in healthy controls (0.46±0.016 and 0.58±0.021 U, resp.). Further electrophoretic studies showed that fraction 3 of cholinesterase was missing in 35.5% of the patients, mostly those with the A(II) blood group. These findings confirm that there are 2 types of peptic ulcer: one related to the genetic factors (patients with the O(I) blood group, nonsecretors with certain types of alkaline phosphatase and pseudocholinesterase and another associated with phenotype.

4494 PERSISTENT INHIBITION OF GASTRIC SECRETION BY AN INTRAVENOUS 12-HOUR INFUSION OF CALCITONIN IN NORMALS, PEPTIC ULCER AND HIGH RISK PATIENTS. *(E.)* Hotz, J.; Goebell, H.; Minne, H.;

Ziegler, R. (Cent. Intern. Med. Pediatr., Univ. Ulm, Germany). *Digestion* 11(3/4):311-314, 1974.

The effect of a 12-hr infusion of thyrocalcitonin (20-60 MRC U/hr) on unstimulated acid and pepsin secretion was studied in 4 healthy volunteers, 3 peptic ulcer patients and 3 high risk patients. The high-risk patients suffered from cerebral ischemic syndrome, pulmonary decompensation, and subarachnodial hemorrhage, resp. A striking reduction of total acid output took place in 3 normal subjects and in 2 peptic ulcer patients compared with control experiments. All high-risk patients showed a marked suppression of acidity during and after thyrocalcitonin infusion (40 MRC U/hr). Pepsin secretion paralleled acid output in all cases. In 4/7 cases, side effects (slight nausea and transient headache) occurred. The results suggest that the therapeutic application of thyrocalcitonin by an i.v. route may be restricted to selected cases.

4495 RESULTS OF STIMULATION OF THE STOMACH WITH HISTALOG IN 32 NEGROES WITH CHRONIC DUODENAL ULCER. *(E.)* Lutete-Kelani, M. (Clin. Intern. Dis., Natl. Univ. Lubumbashi, Zire). *Mater. Med. Pol.* 6(2):136-139, 1974.

Gastric secretion after betazole was studied in Africans suffering from duodenal ulcer, and the values were compared with normal values from Europeans. Basal and betazole-stimulated (2 mg/kg, s.c.) gastric secretion was collected in 15-min portions during 90 min from 32 patients (26 men and 6 women) with chronic duodenal ulcer. Basal gastric secretion (2.45 ± 2.3 mEg/liter in men and 0.69 in women) did not exceed the normal values for Europeans. Gastric secretion stimulated with betazole in the same group of patients (17 ± 10.91 mEg/liter in men and 8.6 in women) was within normal values. The data suggest that hypersecretion of gastric juice is not an essential factor in the pathogenesis of duodenal ulcer in black Africans.

4496 EFFECTS OF AN ANTIULCER AGENT, N-ACETYL-L-GLUTAMINE ALUMINUM COMPLEX (KW-110) ON THE DUODENAL AND GASTRIC ULCER MODELS IN THE RAT. *(E.)* Takagi, K.; Takeuchi, K.; Nakamura, K.; Morita, A.; Okabe, S. (Fac. Pharm. Sci., Univ. Tokyo, Japan). *Jpn. J. Pharmacol.* 24(3):357-361, 1974.

The effects of N-acetyl-L-glutamine aluminum complex (KW-110) and atropine sulfate on duodenal and gastric ulcers were compared in studies on male Donryu rats. KW-110 (p.o. for 8 days) significantly accelerated the healing of duodenal ulcers at 1000 mg/kg/day (38.0%) or 3000 mg/kg/day (40.0%). The effect of atropine sulfate (20 mg/kg/day for 8 days, s.c.) was not significant. Although atropine sulfate (10 mg/kg) completely inhibited stress ulcer formation, KW-110 was also strongly inhibitory at 500 mg/kg (66.0%) or 1000 mg/kg (81.7%). At 500 and 1000 mg/kg, KW-110 inhibited Shay ulceration by 44.3% and 100%, resp., and aspirin-induced lesions by 32.8% and 69.1%, resp. At 10 mg/kg, atropine sulfate inhibited Shay ulceration and aspirin-induced lesions by 74.4% and 33.8%, resp. Prevention of Shay ulceration by KW-110 appeared to be caused by raised pH value and reduced pepsin activity. These results confirm that KW-110 is a promising antiulcer agent in experimental gastrointestinal ulcer models.

4497 CONTROLLED STUDY OF THE SURGICAL TREATMENT OF DUODENAL ULCER. *(E.)* Dwight, R. W.; Schimmel, E. M.; O'Hara, E. T.; Hechtman, H. B.; Williams, L. F.; Vollman, R. W.; Maselli, J. P.; Johnson, W. C.; Nabseth, D. C. (Boston VA Hosp., Mass.). *Am. J. Surg.* 129(4):374-379, 1975.

Vagotomy and drainage was compared with vagotomy and hemigastrectomy in treatment of duodenal ulcer. Of 100 male veterans, 51 were selected for hemigastrectomies and 49 for drainage procedures on a random basis. Of the hemigastrectomies, 18 were Billroth I and 33 Billroth II operations; of the drainage procedures 28 were Heinke-Mikulicz, 5 Jaboulay, 8 Finney, 2 Wagensteen pyloroplasties, and 6 posterior gastroenterostomies. Both patient groups were comparable in age, duration of symptoms, and preoperative peak acid output. Every 3 months in the 1st yr after surgery, and then every 6 months, each patient was weighed and given a questionnaire about work, bowel and eating habits, symptoms, and use of medication. The abdomen was examined for tenderness, succussion, solidity of the scar; blood was tested for serum iron, iron-binding capacity, and hematocrit; and stools were given a guaiac test. Surgical results were considered excellent when patient had no complaints; good when there were mild symptoms; and poor when symptoms were marked enough to affect the patient's way of life; and a failure when ulcer recurred, reoperation was necessary, or incapacitating dumping and malnutrition occurred. Follow-up was 5 yr or more in 52 patients, 3 yr in 80, and at least 1 yr in the remainder, excluding 3 who were not followed up. Operating time was significantly longer for hemigastrectomy (245 min) than for drainage (170 min) and transfusion was required in 15 of the former and 4 of the latter. Six major complications and five minor ones occurred after resection, while 2 major and 8 minor complications occurred after drainage. After resection, results were excellent in 45%, good in 35%, poor in 9% and failure in 8%. After drainage, the corresponding results were 58%, 25%, 8%, and 8%, resp. Failures after resection included 2 recurrent ulcers, 1 reoperation for stomal obstruction, and 1 case of severe dumping and malnutrition. Failures after drainage included 3 recurrent ulcers, 1 reoperation for pyloric obstruction. The type of drainage procedure or anastomosis used did not affect the results. Blood tests showed no significant differences between the two groups. Mean weight loss was significantly greater after resection (17.5 pounds) than drainage (10.5 pounds). It is concluded that there is no significant difference on mortality or recurrence of ulcers between vagotomy and resection and vagotomy and drainage; however, as nutrition is maintained better after drainage, this procedure is recommended.

4498 GASTRO-DUODENAL ULCER IN KIDNEY-TRANSPLANTED PATIENTS RECEIVING IMMUNOSUPPRESSIVE TREATMENT. *(E.)* Rasmussen, K.; Christiansen, J.; Nielsen, O. V.; McNair, A.; Sorenson, M. B. (Glostrup Hosp., Denmark). *Acta Chir. Scan.* 141(1):61-64, 1975.

The clinical course was evaluated in 14 cases of gastroduodenal ulcer in kidney-transplanted patients receiving immunosuppressive therapy. Of 229 patients who received kidney transplants in the Copenhagen hospitals between 1968 and 1973, 14 had or developed peptic ulcers requiring treatment in connection with the transplantation. In three patients a prophylactic gastric operation was carried out prior to the kidney transplantation; in two with ulcer dyspepsia during the uremic period, conservative treatment with antacids and controlled diet was given prior to transplantation; conservative treatment was also given in three cases of peptic ulcer complications during immunosuppressive treatment; and surgery was performed in five patients with peptic ulcer complications during immunosuppressive treatment. Among the eight patients in whom bleeding peptic ulcer occurred during immunosuppressive therapy, six died due to hemmorrhage. Five were operated acutely, three of whom died. In three in whom the ulcer was neither removed nor excluded, hemmorrhage from the same ulcer recurred during immunosuppressive treatment. Of the patients treated with prophylactic surgery prior to kidney transplantation, only one showed symptons of transient dyspepsia during immunosuppressive therapy. The results indicate that a verified peptic ulcer occurring prior to transplantation should be subjected to prophylactic surgery. In the case of ulcer dyspepsia arising during immunosuppressive treatment, the treatment should be elective surgery and discontinuation of the immunosuppressive drugs during the healing period. Radiological examination of the stomach and esophagogastroduodenoscopy should be a standard procedure prior to kidney transplantation.

4499 EFFECT OF ATROPINE ON GASTRIC SECRETION OF ACID, PEPSIN, AND ELECTROLYTES ELICITED BY MAXIMAL STIMULATION WITH HISTAMINE AND PENTAGASTRIN IN NORMAL CONTROL AND DUODENAL ULCER PATIENTS FROM BOMBAY. *(E.)* Pimparkar, B. D.; Bhiwankar, N. T.; Kulharni, B. S.; Mehta, J. M. (K. E. M. Hosp., Bombay, India). *Indian J. Med. Res.* 62(11):1736-1750, 1974.

To determine whether gastric acid secretion stimulated by augmented doses of histamine and pentagastrin could be suppressed by atropine, an augmented histamine test (76 µg/kg histamine acid phosphate) and a pentagastrin test (6 µg/kg) were carried out in 10 normal controls and 15 patients with duodenal ulcer. Atropine (1.2 mg) was given twice, once during the basal period and again after stimulation with histamine and pentagastrin. In both controls and patients, atropine significantly suppressed basal and stimulated acid secretion. During the basal period and after stimulation with histamine or pentagastrin, atropine resulted in identical suppression in gastric pepsin, sodium, potassium, and chloride in both groups. With atropine there was significant suppression in volume but no significant changes in the concentration of acid pepsin and electrolytes. It is concluded that histamine and pentagastrin act through the vagus since their action after augmented doses is suppressed by atropine. The predominant action of atropine is a decrease in gastric secretory volume which reflexly limits the output of various ingredients, even though there is no significant change in their concentrations.

4500 STUDIES ON THE HEALING OF EXPERIMENTAL ULCER IN THE RAT II: INFLUENCE OF ANTI-INFLAMMATORY DRUGS ON THE HEALING OF THE ACETIC ACID ULCER AND THE COMPONENTS IN GASTRIC TISSUE. *(E.)* Takagi, K.; Abe, Y. (Fac. Pharm. Sci., Univ. Tokyo, Japan). *Jpn. J. Pharmacol.* 24(3):345-356, 1974.

The relation between chemical components of an acetic acid ulcer site and ulcer healing was investigated in male Donryu rats together with the effects of antiinflammatory drugs on the ulcer. The hexosamine content in the corpus of normal rat stomach decreased when sodium salicylate (0.5%) or phenylbutazone (0.2%) was added to the diet for 20 days. Both sodium salicylate and phenylbutazone delayed the healing of 10% acetic acid ulcer, and resulted in a simultaneous decrease in hexosamine at the ulcer site. Prednisolone (25 mg/kg s.c.) and cortisone (50 mg/kg s.c.) retarded ulcer healing in the early stages of administration, when hexosamine and hydroxyproline decreased significantly. Sodium salicylate, by contrast, delayed ulcer healing in the early and also later stages of administration. A decrease in hexosamine content in the injured mucosa and a decrease in hydroxyproline content in the injured stroma paralleled an increase of area index and grade index, resp. These results suggest that the mucosal hexosamine and stromal hydroxyproline content in the injured portion play an important role in ulcer healing. The data also demonstrate differences between steroidal and nonsteroidal anti-inflammatory agents with respect to retardation of ulcer healing.

4501 CYSTEAMINE-INDUCED DUODENAL ULCERS: A NEW MODEL TO TEST ANTIULCER AGENTS. *(E.)* Robert, A.; Nezamis, J. E.; Lancaster, C.; Badalamenti, J. N. (Upjohn Co., Kalamazoo, Mich.). *Digestion* 11(3/4):199-214, 1974.

The effect of various agents on cysteamine-induced ulcers and the effect of cysteamine on gastric secretion were studied in Upjohn rats to evaluate the procedure as an assay for antiulcer agents. Cysteamine was administered at doses 230-465 mg/kg, s.c. and 460-900 mg/kg, p.o.; the duodeno-ulcerogenic property of cysteamine was dose-dependent after either route of administration. Duodenal ulcers were produced within 24 hr; at least 50% of the ulcers perforated with 425 mg/kg, s.c. Cysteamine-induced duodenal ulcers were prevented by an anticholinergic agent (methscopolamine bromide), by an antacid (Malcogel) by prostaglandins (PGE_2 and 16,16-dimethyl PGE_2), by prednisolone, and by interruption of the gastroduodenal transit (pylorus ligation). Fasting

did not affect the incidence but reduced the severity of the ulcers. Incidence and severity were higher in female than in male rats. Deoxycorticosterone and ACTH did not affect the ulcers. Gastric secretion was inhibited by cysteamine and was dose-dependent; however, some acid secretion and the passage of acid gastric contents over the duodenum were required for cysteamine to produce ulcers. The results indicate that cysteamine-induced duodenal ulcers provide an excellent model for the study of antiulcer agents.

4502 EFFECT OF METIAMIDE ON HISTAMINE METABOLISM IN MAN. *(E.)* Shepherd, D. M.; Thjodleifsson, B.; Turnbull, M. J.; Wormsley, K. G. (Dep. Pharmacol. Ther., Univ. Dundee, Scotland). *Digestion* 11(3/4):307-310, 1974.

The effect of metiamide on the urinary excretion of histamine, 1,4-methylhistamine, and 5-hydroxyindol-3-yl-acetic acid was studied in 12 duodenal ulcer patients (aged 22-65 yr) receiving metiamide therapy (600-1200 mg/day). Administration of metiamide resulted in an increased excretion of 1,4-methylhistamine (263 to 465 µg/24 hr) and a decreased secretion of histamine (29.5 to 19 µg/24 hr). The excretion of 5-hydroxyindol-3-yl-acetic acid was not significantly affected. Blood metiamide levels were 1 to 6 x 10^{-6}M and the urinary amine excretion pattern was compatible with an *in vivo* stimulation of histamine methyltransferase (HMT) activity. If histamine is involved in mediating the gastric secretory response to stimulants in man, then the stimulation of HTM activity by metiamide may contribute to the inhibition of gastric secretion produced by the drug.

4503 GLYCOPROTEINS IN THE STOMACH OF PATIENTS WITH GASTRIC AND DUODENAL ULCERS: PATHOPHYSIOLOGICAL FINDINGS AND EFFECT OF TREATMENT. *(Cz.)* Domschke, W.; Demling, L. (Clin. Intern. Med., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Vnitr. Lek.* 20(7):625-630, 1974.

The rate of gastric acid secretion was measured before and for 1.5 hr after pentagastrin infusion (1.5 g/kg/hr i.v.), as was the output of glycoproteins containing *N*-acetylneuraminic acid (NANA), in 22 patients with gastric ulcers, 20 with duodenal ulcers, and in 45 normal controls. These studies were performed on patients before and 4 weeks after carbenoxalone treatment. Patients with gastric ulcers were given 300 mg/day carbenoxalone sodium for 1 week and then 150 mg/day, while patients with duodenal ulcers received 300 mg/day of the enteric-coated form of the drug. In normal subjects there was a very good linear relation between the peak acid output and the production of glycoproteins containing NANA. This relation was also evident in patients with gastric ulcers, but the rate of HCl production was greater than in controls. No such relation was found between these two parameters in patients with duodenal ulcers. After 4 weeks of treatment with carbenoxalone sodium, the output of glycoproteins containing NANA increased significantly and even exceeded normal values in patients with gastric ulcers. However, carbenoxalone sodium did not stimulate mucus production in the stomach of patients with duodenal ulcers since the drug was not absorbed until after it had passed through the stomach.

4504 CALCULATED MAXIMAL RESPONSE AND ED_{50} FOR ACID SECRETION IN CONTROL SUBJECTS AND PATIENTS WITH DUODENAL ULCER. *(E.)* Desai, H. G.; Zaveri, M. P.; Antia, F. P. (B. Y. L. Nair Charitable Hosp., Bombay, India). *Indian J. Med. Res.* 62(12):1843-1849, 1974.

Values of calculated maximal response (CMR) and ED_{50} on i.v. histamine tests were determined in 13 control subjects in India and 20 male patients with duodenal ulcer. Dose-response curves were obtained using 0.4-2.8 mg/kg doses of histamine acid phosphate. The mean ED_{50} value was lower in ulcer patients (9.7 ± 5.3 µg/kg/hr) than in controls (12.5 ± 5.3), but the difference was not significant. CMR values in controls (8.0 ± 2.3 mEq/155 min) and in patients (10.1 ± 3.1) were significantly different, as were the highest observed values (HOV) of acid output (6.3 ± 1.5 mEq/15 min in controls and 8.5 ± 2.7 in patients). However, the lowest HOV value (2.5 mEq/15 min and the lowest CMR value (3.5 mEq/15 min) were observed in duodenal ulcer patients. The value of ED_{50} in relation to body weight showed a significant correlation with the body weight of controls but not of ulcer patients. The ED_{50} vlaues obtained in this study differ from those reported for North American patients with duodenal ulcer; the difference may result from marked differences in maximal acid output in the patients from these countries.

4505 SECRETION OF GASTRIC LIPASE, PEPSIN AND HYDROGEN IONS FOLLOWING PENTAGASTRIN ADMINISTRATION IN PATIENTS WITH DUODENAL ULCER. *(Pol.)* Trompeter, G.; Komarowska, M.; Szafran, Z.; Popiela, T. (Med. Acad., Krakow, Poland). *Przegl. Lek.* 31(2): 287-293, 1974.

Pentagastrin was administered for 3 hr by continuous i.v. infusion to six male patients (aged 20-43 yr) with duodenal ulcer. The dose was increased every 30 min (8, 64, 256, 1024, 2048, and 8192 ng/kg), gastric juice collected every 10 min and hydrogen ion concentration as well as lipase and pepsin activity determined in each of the 18 fractions. In fasting gastric juice the hydrogen ion concentration varied between 10-61 mEq/liter, activity of lipase between 0.67-4.61 U/min/ml, of pepsin 0.18-0.56 x 10^{-2} Anson U/min/ml. Highest lipase activity was obtained in fraction collected after 140-150 min, the highest pepsin activity 70-80 min, highest ion concentration 130-140 min of test. Despite a great variation in values, statistical analysis revealed a highly significant positive correlation between all three measured parameters. The most significant correlation coefficient was obtained between hydrogen ion concentration and lipase activity. In calculations taking into account the output values a high correlation coefficient was obtained for output of both enzymes. It is concluded that pentagastrin stimulation caused active secretion not only

of pepsin and hydrogen ions but also of gastric lipase.

4506 STUDIES ON THE BIOELECTRICAL ACTIVITY OF THE STOMACH IN PEPTIC ULCER. *(Pol.)* Wryzykowski, R.; Szczgiel, B.; Rucinski, J. (Med. Acad., Warsaw, Poland). *Przegl. Lek.* 31(2):283-286, 1974.

External electrography using the EMA electrogastrograph Model EGS-4M was performed on 21 healthy volunteers, 10 patients with gastric, 14 with duodenal ulcer and six with narrowing of the pylorus. The tests were performed after overnight fasting and in the control group also after food, atropine or prostygmine stimulation. The time of recordings varied between 10-60 min. The electrogastrograms were interpreted by calculating the average frequency of biopotential changes/min and average amplitude in mV. It was found that in comparison to controls (average frequency/min = 3.5, average ampliture/mV = 0.3) the basal electrical rhythm was decreased in patients with gastric ulcer in regard to average frequency (1.1) and average amplitude (0.1); increased in regard to average frequency (4.5) without change in amplitude in patients with duodenal ulcer, while in patients with narrowing of the pylorus and ulcer a significant increase in amplitude (0.8) without change in frequency was observed. Both frequency and amplitude increased. Atropine (0.5 mg) caused a decrease in amplitude and slowing down of frequency. Prostigmin caused a significant increase in the bioelectric activity of the stomach both in regard to amplitude and frequency.

4507 EFFECT OF CALCIUM AND HISTAMINE ON GASTRIC ACID SECRETION IN MAN. *(E.)* Dalal, N. J.; Vakil, B. J.; Gangrade, R. R.; Bhise, K. B.; Shah, P. N. (J. J. Group Hosp., Bombay, India). *Indian J. Med. Res.* 62(11):1652-1658, 1974.

Gastric acid secretion in response to calcium infusion and subsequent histamine injection was studied in seven Indian patients with duodenal ulcer, six patients with hyperacidity, and seven normal subjects. Calcium infusion (4 mg/kg/hr, i.v., for 3 hr) resulted in a significant rise in gastric acid secretion over basal values; the calcium-stimulated maximum acid output was 2.19 times the basal output in controls, 2.23 times the basal output in duodenal ulcer cases, and 2.84 times the basal output in hyperacidity cases. Histamine administration (0.04 mg/kg s.c.) induced a further rise in acid secretion. Maximum acid secretion after calcium infusion was 63% and 55%, resp., of post-histamine values in patients with duodenal ulcer and those with hyperacidity; in controls the post-calcium value was 41% of the post-histamine value. The difference in the response between controls and the duodenal ulcer and hyperacidity cases was significant, whereas the difference in the response between the two groups of patients was not significant. To determine if there is any potentiation between calcium and histamine, four controls were infused with histamine (0.04 mg/kg/hr) until a steady state of gastric acid secretion was reached; calcium was then given simultaneously with histamine for 2 1/2 hr. No further increase in gastric acid secretion occurred in response to the calcium infusion. The absence of potentiation between histamine and calcium indicates that calcium provides a submaximal stimulus for acid secretion compared with histamine.

4508 HIGHLY SELECTIVE VAGOTOMY FOR DUODENAL ULCER: DO HYPERSECRETORS NEED ANTRECTOMY? *(E.)* Johnston, D.; Pickford, I. R.; Walker, B. E.; Goligher, J. C. (Gen. Infirm., Leeds, England). *Br. Med. J.* 1(5960):716-718, 1975.

Patients who had undergone highly selective vagotomy for duodenal ulcer were reviewed 2-6 yr later to determine if the surgical treatment was adequate or if antrectomy was also needed in hypersecretors. The clinical results in 40 hypersecretors were compared with those in 60 patients with normal acid secretion. The overall clinical results were similar in both groups; the incidence of pain of ulcer type was 6% in each group, peak acid output was similar in both groups, and the incidence of incomplete vagotomy was 7% in each group. In no case was a recurrent ulcer seen. The incidence of individual symptoms was about equal in the two groups, however early dumping was recorded in 1% of hypersecretors compared with 6% of normal secretors. The results suggest that the addition of antrectomy to vagotomy is unnecessary when patients with hypersecretion are treated by highly selective vagotomy, providing the vagotomy is complete.

4509 MARGINAL ULCER IN ACHLORHYDRIC PATIENTS. *(E.)* Tauxe, R. V.; Wright, L. F.; Hirschowitz, B. I. (Dep. Med., Univ. Alabama, Birmingham). *Ann. Surg.* 181(4):455-457, 1975.

Details of three cases of endoscopically proven anastomotic ulceration were reported in patients with histamine-fast achlorhydria. Basal secretion was collected in 15-min samples for 1 hr; volume and pH determinations were made. Histamine acid phosphate (0.04 mg/kg) was injected s.c. after basal secretion collection and 20 mg benadryl was also injected to minimize side effects. Two cases demonstrated anemia and two had pernicious anemia. Achlorhydria was present even in the two who had not undergone vagotomy. All three patients had a history of alcohol abuse, and one abused aspirin as well. The cases demonstrated that achlorhydria did not protect against anastomotic ulceration and that it may be erroneous to consider all anastomotic ulceration as a continuation of the original peptic disease. The results suggest that stomal ulceration is frequently a multifactorial disease which occurs because the gastrojejunal stoma has less resistance to damage than does the adjacent unaltered gastric and jejunal mucosa.

4510 A CONTROLLED, RANDOMIZED TRIAL OF HIGHLY SELECTIVE VAGOTOMY VERSUS SELECTIVE VAGOTOMY AND PYLOROPLASTY IN THE TREATMENT OF DUODENAL ULCER. *(E.)* Kronborg, O.; Madsen, P. (Bispebjerg Hosp., Copenhagen, Denmark). *Gut* 16(4):268-271, 1975.

The results of highly selective vagotomy without drainage and selective vagotomy with pyloroplasty for duodenal ulcer were compared in a randomized, controlled trial of a series of 100 patients. Fifty patients received one method of treatment and 50 the other. The clinical results were obtained 1 yr after operation. The frequency of dumping, diarrhea, and epigastric fullness was significantly lower after highly selective (6, 6, and 8%) than after selective vagotomy (30, 20, and 28%). Recurrent and persisting duodenal ulcers appearing 1-4 yr after operations were significantly more frequent after highly selective (22%) than after selective vagotomy (8%). No significant relations were found between recurrent ulceration and gastric acid secretion measurements after the two operations. The Hollander response was early positive in 28% and late positive in 30% of the patients subjected to highly selective vagotomy, while the corresponding figures after selective vagotomy were 26 and 32%, resp. The results suggest that the low frequency of dumping and diarrhea after highly selective vagotomy is offset by a high recurrence rate, and according to Visick grading, a complementary relation exists between recurrence and postvagotomy symptoms in surgery for duodenal ulcer.

4511 GASTRIC-ACID OUTPUT, PEPSIN OUTPUT, AND LEAN BODY MASS IN NORMAL AND DUODENAL-ULCER SUBJECTS. *(E.)* Elder, J. B.; Smith, I. S. (R. Infirm., Manchester, England). *Lancet* 1(7914): 1000-1003, 1975.

The relation of pepsin output to gastric acid output and to lean body mass were investigated in 13 normal subjects and 40 duodenal ulcer patients. Peak pepsin output was stimulated by insulin-induced hypoglycemia and the enzyme was assayed by Piper's method. Peak acid output was determined following pentagastrin (6 µg/kg, s.c.). Pepsin output correlated significantly with acid output in normal subjects, and also showed good correlation with lean body mass. In the duodenal ulcer patients, there was no significant overall correlation of pepsin output with acid output or with lean body mass; however, based on the ratios of the three variables, the ulcer patients were separated in two groups whose ratios appeared to correlate with their symptomatic state at time of testing: pepsin output/mEq acid/kg lean body mass was significantly greater for patients in relapse than for patients in remission and for the normal group. The results suggest that both acid and pepsin may be important factors in inducing relapse in duodenal ulcer patients, and that pepsin output studies may prove helpful in understanding pathogenesis of relapse.

4512 SULFHYDRYL COMPOUNDS OF THE GASTRIC MUCOSA IN PATIENTS WITH PEPTIC ULCER. *(Rus.)* Samson, E. I.; Gaidichuk, S. T. (Chernovitsy Med. Inst., USSR). *Vrach. Delo* (1):12-15, 1975.

Sulfhydryl compounds were studied in 35 patients with peptic ulcer at the time of acute and moderate aggravation of the disease. Nine patients had normal structure of the mucosa; 24 had surface gastritis; in two patients peptic glands were affected but there was no evidence of atrophy. Acidity was elevated in 28 (pH below 1.5); normal in six (pH = 1.5 - 2.4); and low in one. It was demonstrated that in cases with normal mucosa and surface gastritis, sulfhydryl compounds were present in the basal membrane of surface epithelium and gastric fossae. The apical areas of both the surface epithelium and fossae were free from sulfhydryl compounds. In patients with marked morphological changes in the mucosa, sulfhydryl compounds were observed in the surface epithelium. There was evidence of faint staining in the epithelia of basal membrane and gastric fossae. A correlation was established between the intensity of staining of the parietal cells and elevated acidity. It is concluded that the distribution of sulfhydryl compounds in gastric mucosa of peptic ulcer patients correlates with the type of concomitant gastritis. It is suggested that sulfhydryl compounds may be involved in acid production.

4513 GASTRIC AND DUODENAL ULCERS. *(Fr.)* Lambert, R. (Med. Fac., Lyon, France). *Recherche* 5(45):436-446, 1974.

4514 RECENT INFORMATION ABOUT THE PATHOGENESIS OF STOMACH ULCERS AND ITS IMPLICATIONS FOR THERAPY. *(Ger.)* Rothmund, M. (Surg. Clin., Univ. Mainz, Germany). *Therapiewoche* 24(39):4221-4222, 4224, 4227, 1974.

4515 PATHOGENESIS AND AETIOLOGY OF ULCER DISEASE. PART I: GASTRIC ULCER. *(E.)* Blum, A. L.; Peter, P.; Krejs, G. J. (Triemli Munic. Hosp., Switzerland). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):47-54, 1975.

4516 COMMON GASTROENTEROLOGICAL PROBLEMS. III: CHRONIC BENIGN GASTRIC ULCER. *(E.)* Salter, R. H. (Cumberland Infirm., Carlisle, England). *Practitioner* 214(1281):407-411, 1975.

4517 ASSESSMENT AND TREATMENT OF RECURRENT PEPTIC ULCERATION. *(E.)* Steinberg, D. M.; Masselink, B. A.; Alexander-Williams, J. (Gen. Hsop., Birmingham, England). *Ann. R. Coll. Surg. Engl.* 56 (3):135-140, 1975.

4518 THERAPEUTIC TRIAL OF PROGLUMIDE IN GASTRODUODENAL DISEASES. *(Fr.)* Brandt, C.; Peter, B.; Stahl, A.; Bockel, R. (Hosp. Cent., Univ. Strasbourg, France). *J. Med. Strasbourg* 5(6/7):493-496, 1974.

4519 AN ASSESSMENT OF THE REPRODUCIBILITY AND SAFETY OF 2-DEOXY-D-GLUCOSE AS A GASTRIC ACID STIMULANT IN DUODENAL ULCER PATIENTS. *(E.)* Gough, A. L.; Keddie, N. C. (Manchester R. Infirm., England). *Gut* 16(3):171-176, 1975.

4520 INTRAGASTRIC TELEMETRIC EVALUATION OF THE EFFICACY AND DURATION OF ACTION OF VARIOUS BUFFER SUBSTANCES AND AN ANTICHOLINERGIC DRUG ADMINISTERED ORALLY. *(E.)* Plessier, J.; Economopoulos, P.; Kusha, I.; Chehab, A. (St. Antione Hosp., Paris, France). *Arzneim. Forsch.* 24(12):2051-2053, 1974.

4521 COMPARISON OF THE EFFECT OF VAGOLYTIC AGENTS AND VAGOTOMY ON THE ELECTROGASTROENTEROGRAPHS OF FASTING PATIENTS WITH DUODENAL ULCERS. *(Fr.)* Bertrand, J.; Thillier, J. L. (Univ. Hosp. Cent., Tours, France). *Biol. Gastroenterol. (Paris)* 7(4):292, 1974.

4522 TRANSTHORACIC VAGOTOMY IN TREATING PEPTIC ULCERS IN THE JEJUNUM. *(Ger.)* Grotelueschen, B.; Reichel, K.; Pichlmayr, R. (Med. Coll., Hannover, Germany). *Chirurg* 45(10):462-464, 1974.

4523 SELECTIVE PROXIMAL VAGOTOMY WITHOUT DRAINAGE OPERATION. *(Hun.)* Berger, R. (Dombovar City Hosp., Hungary). *Magy. Sebesz.* 27(5):285-287, 1974.

4524 PHYSIOLOGICAL AND CLINICAL SIGNIFICANCE OF HIGHLY SELECTIVE VAGOTOMY WITHOUT A DRAINAGE PROCEDURE. *(E.)* Johnston, D. (Gen. Infirm., Leeds, England). *Digestion* 11(3/4):275-288, 1974.

4525 HIGHLY SELECTIVE VAGOTOMY FOR DUODENAL ULCER: IS ANTRECTOMY NEEDED FOR THE HYPERSECRETORS? *(E.)* Pickford, I. R.; Johnston, D.; Walker, B. E.; Goligher, J. C. (Gen. Infirm., Leeds, England). *Br. J. Surg.* 62(2):152-153, 1975.

4526 THE EFFECT OF HIGHLY SELECTIVE VAGOTOMY AND TRUNCAL VAGOTOMY WITH PYLOROPLASTY ON GASTRIC EMPTYING OF SOLID FOOD IN MAN. *(E.)* Sheiner, H. J.; Duthie, H. L.; Barber, D. C.; Howlett, P. J. (Sheffield R. Infirm., England). *Br. J. Surg.* 62(2):163-164, 1975.

4527 VAGOTOMY ASSOCIATED WITH PYLOROPLASTY: RADIOLOGICAL STUDY. *(Por.)* de Carvalho Whitaker, J. de C.; Chacon, J. P.; Magalhaes, A. de A. (Paulist Med. Sch., Sao Paulo, Brazil). *An. Pau Paulistas Med. Chir.* 102(6):59-70, 1975.

4528 THIRD WALTER B. CANNON LECTURE, 1974. THE SURGICAL TREATMENT OF GASTROINTESTINAL ULCER: PAST AND FUTURE. *(E.)* Dunphy, J. E. (Sch. Med., Univ. California, San Francisco). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):229-235, 1975.

4529 OPERATIVE MORTALITY AND POSTOPERATIVE MORBIDITY OF HIGHLY SELECTIVE VAGOTOMY. *(E.)* Johnston, D. (Gen. Inform., Leeds, England). *Br. J. Surg.* 62(2):160, 1975.

4530 THE IMMEDIATE AND DELAYED EFFECTS OF DIFFERENT TYPES OF VAGOTOMY ON HUMAN GASTRIC MYOELECTRICAL ACTIVITY. *(E.)* Stoddard, C. J.; Smallwood, R.; Brown, B. N.; Duthie, H. L. (Sheffield R. Infirm., England). *Gut* 16(3):165-170, 1975.

4531 MUCOSA OF THE RESECTED STOMACH - HISTOTOPOGRAPHIC AND FUNCTIONAL INVESTIGATIONS. *(E.)* Stadelmann, O.; Miederer, S. E.; Elster, K. (Med. Polyclin., Bonn, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):61, 1975.

4532 UNUSUAL PERFORATION OF A DUODENAL ULCER. *(Ger.)* Gunther, R.; Benken, U.; Schier, J. (Inst. Clin. Radiol., Univ. Mainz, Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 121(2):248-249, 1974.

4533 PEPTIC ULCER AND PULMONARY DISEASE. *(E.)* Viskum, K. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Respir. Dis.* 55(5):284-290, 1974.

4534 RELATIONS BETWEEN PEAK STIMULATED GASTRIC SECRETION, BODY WEIGHT, AND ACTIVE BODY MASS IN PATIENTS WITH DUODENAL ULCERS. *(Ger.)* Jablonska, M.; Fucik, M.; Eiselt, E.; Malkovska, K. (4th Med. Clin., Charles Univ., Prague, Czechoslovakia). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):215-217, 1974.

4535 BLEEDING CAUSED BY ACUTE PEPTIC ULCERATION. *(Fr.)* Chigot, J. P. (Hosp. de la Pitie, Paris, France). *Rev. Fr. Gastroenterol.* (103):35-38, 41-42, 44, 1974.

4536 THE EFFECT OF ALMAGEL ON THE EXCRETION OF NEUTRAL 17-KETOSTEROIDS, DEHYDROISOANDROSTERONE AND FREE CATECHOLAMINES IN THE URINE OF PATIENTS WITH PEPTIC ULCER. *(Rus.)* Shevchenko, B. I. (Chernovitsy Med. Inst., USSR). *Vrach. Delo.* (3):65-69, 1975.

4537 FLUCTUATIONS IN THE SKIN AND TEMPERATURE SENSITIVITY OF HEAD'S ZONES AND THEIR ROLE IN THERMOTHERAPY OF PEPTIC ULCER AND CHRONIC CHOLECYSTITIS. *(Rus.)* Solomianskii, A. E. (Dagestan Med. Inst., Makhachkala, USSR). *Vorp. Kurortol. Fizioter. Lech. Fiz. Kult.* (5):439-442, 1974.

4538 EFFECT OF SYMPATHECTOMY INDUCED BY GUANETHIDINE ON THE ULCEROGENIC ACTION OF PHENYLBUTAZONE IN RATS OF VARIOUS AGES. *(Sl.)* Kohut, A.; Nicak, A. (Med. Fac., Sararik Univ., Kosice, Czechoslovakia). *Cesk. Gastroenterol. Vyz.* 28(8):529-531, 1974.

4539 THE ULCEROGENIC EFFECT OF BILE AND BILE ACIDS IN RATS SUBJECTED TO IMMOBILIZATION STRESS. *(Ger.)* Wiesener, J. (Pathol. Inst., Free Univ., Berlin, Germany). *Z. Gastroenterol.* 12(7): 539-546, 1974.

See also, 4182, 4215, 4219, 4226, 4227, 4228, 4302, 4308, 4369, 4415, 4421, 4432, 4436, 4592, 5054, 5062.

SMALL INTESTINE

4540 INTESTINAL ENTEROKINASE DEFICIENCY. OCCURRENCE IN TWO SIBS AND AGE DEPENDENCY OF CLINICAL EXPRESSION. *(E.)* Haworth, J. C.; Hadorn,B.; Gourley, B.; Prasad, A.; Troesch, V. (Health Sci. Cent., Winnipeg, Canada). *Arch. Dis. Child.* 50(4):277-282, 1975.

Intestinal enterokinase deficiency in two siblings is described. The parents were not related, were healthy and free of gastrointestinal symptoms. A boy (6 weeks old) failed to gain weight and had vomiting, diarrhea, edema, hypoproteinemia, and anemia in early infancy. His duodenal juice contained very low or no proteolytic enzyme activity, which increased markedly after addition of enterokinase. He was treated with a regular diet with pancreatic extract and gained weight rapidly. At 44 months of age, he was normal, apart from some developmental delay, and no longer required pancreatic extract. The boy's older sister, who had similar symptoms in early infancy but then grew normally, had the same abnormality in her duodenal juice when seen at 4 yr. Enterokinase activity was virtually absent in the duodenal mucosa of both patients. Duodenal biopsies showed that the mucosal morphology was normal in both children. It is suggested that enterokinase deficiency is an inherited congenital defect and not the result of mucosal damage. Affected patients may show spontaneous improvement and normal growth after the age of 6-12 months. This phenomenon may be related to the decreasing growth velocity during the first 2 yr of life and the concomitant decrease in protein requirements/U body weight.

4541 INFLAMMATORY PSEUDOTUMORS (INFLAMMATORY FIBROUS POLYPS) OF THE SMALL INTESTINE: A CLINICOPATHOLOGIC STUDY. *(E.)* LiVolsi, V. A.; Perzin, K. H. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Am. J. Dig. Dis.* 20(4):325-336, 1975.

The clinical and pathologic features of 25 inflammatory pseudotumors of the small intestine are reported. For each specimen, pathology protocols, microscopic slides, and clinical records were reviewed. The group included nine males and 15 females (sex was unknown in one case) ranging in age from 6-86 yr (three patients were identified as adults only). In 16 patients, the diagnosis of small-intestinal obstruction, probably due to neoplasm,was made. The other patients were investigated for other reasons. All 25 patients underwent exploratory laparotomy. Sixteen lesions were found in the ileum and two in the jejunum. Intussusception was found associated with nine of 16 ileal lesions, with one of two jejunal tumors and with four cases in which the exact location of the lesion was unspecified. All 22 patients with lesions of the ileum or jejunum were treated by resection of the involved bowel segment. Of three patients with duodenal lesions, one underwent partial gastrectomy, one had a local excision of a duodenal nodule, and one was biopsied *via* a duodenotomy. Histologically, many cell types were noted in all these cases: fibrocytes, histiocytes, lymphocytes, scattered plasma cells, endothelial cells, leukocytes, and eosinophils. Mucosal involvement was found in 22 specimens. The etiology of the intestinal pseudotumor is unknown. No specific symptoms, laboratory tests, or x-rays established the diagnosis preoperatively. Even at laparotomy, the differential diagnoses included various neoplasms; only by histologic examination could the correct diagnosis be made. Following resection of the involved segment of small intestine, prognosis appears excellent. None of the patients for whom follow-up information was available had a recurrence of disease. It is suggested that intestinal pseudotumors may represent an exuberant granulation tissue response to mucosal ulceration.

4542 THERAPEUTIC CONSIDERATIONS REGARDING 75 PERFORATIONS OF THE SMALL INTESTINE. *(Fr.)* Spay, G. (No affil.). *Lyon Chir.* 71(1):34-38, 1975.

During 17 months in Kabul, Afghanistan, 55 cases of ileal perforation were observed including: 31 perforations due to typhoid; eight to *Ascaris*; three to multiple causes which included *Ascaris* in all three, typhoid in two and intestinal constriction of unknown etiology in the third; one case was due to stress; seven were of indeterminate etiology; and five unclassifiable due to incomplete records. Overall, the mortality rate was 37%, excluding three cases treated very recently. The patients ranged in age from 1.5 to 70 yr with most aged from 10-20 yr. The peak seasons for the perforations were in July, the rainy season following the Indian monsoon, and November, the dry period. Excision and suture was practiced in 13 cases, with success in 77%. This seems to be the optimum treatment for younger patients regardless of etiology. Exteriorization was practiced in 12 cases with only 42% success. This technique seems advisable with reservation in serious cases of typhoid in younger patients. Resection and temporary ileostomy was practiced 25 times, with 64% success. In 17 months in Dakar, Senegal, 20 cases of perforations were seen. They included one confirmed case of typhoid; five of multiple etiology with *Ascaris* in all five; nine of indeterminate etiology, of which six were

suspected typhoid; one each with tuberculosis and excessive cortisone treatments; and three unclassifiable cases. The overall mortality was 50%. Simple suture in 13 cases produced 54% success and resection with anastomosis in four cases produced 50% success. Of the verified or strongly suspected cases of typhoid perforation, the mortality in the Kabul cases was 26%, compared with 33% for the Dakar cases. In general, simple suture is the preferred treatment and particularly favors the prognosis when the patient is seen less than a week after the onset of symptoms. Resection with terminal ileostomy should be reserved for the more serious cases.

4543 INTESTINAL LYMPHANGIECTASIA. LONG-TERM RESULTS WITH MCT DIET. *(E.)* Tift, W. L.; Lloyd, J. K. (Inst. Child Health, London, England). *Arch. Dis. Child.* 50(4):269-276, 1975.

The clinical course of six children with primary intestinal lymphangiectasia who were treated with low fat medium chain triglyceride-supplemented diets for 3-8 yr (four for longer than 5 yr) is described. All children showed rapid and sustained improvement in dependent edema; the asymmetrical edema, resulting from peripheral lymphatic anomalies, was unaffected by dietary and diuretic therapy. The low lymphocyte counts and reduced serum concentrations of albumin and IgG continued on the medium chain triglyceride diet. Laboratory tests indicated continuing chyle leak. Deliberate attempts to relax the restriction of dietary long chain fats were made in four patients. In three, diarrhea consistently recurred within 2-3 weeks of the introduction of a modest amount of long chain fat; two children also developed dependent edema and one ascites. In all three, symptoms subsided with the return to the medium chain triglyceride diet. It is claimed that laboratory evidence of continuing chyle leak does not indicate that treatment has no effect; return of biochemical findings to normal should suggest re-evaluation of the patient to determine whether the chyle leak has ceased, as occurred in one of six children in this group. For the majority of patients, however, it is likely that excessive gastrointestinal protein loss will continue. It is concluded that if such children are to achieve their optimum growth rate, it is necessary to continue a strict low-fat diet, supplemented with medium-chain triglycerides, at least until after puberty.

4544 INTESTINAL LYMPHANGIECTASIA: A REAPPRAISAL. *(E.)* Vardy, P. A.; Lebenthal, E.; Scwachman, H. (Child. Hosp. Med. Cent., Boston, Mass.). *Pediatrics* 55(6):842-852, 1975.

Fifteen children (nine boys and six girls) seen between 1960-1974 with histologically proven intestinal lymphangiectasia (IL) were analyzed retrospectively by clinical, laboratory, radiologic, and histologic criteria. Remissions occurred in most patients and none died. Exacerbations occurred in five children. Diarrhea was present in 14 cases and 13 patients presented before the age of 3 yr. Vomiting occurred in eight patients and growth retardation in seven. Four children had associated peripheral lymphedema and two of these had a family history of lymphedema; both had affected fathers and one had affected siblings and paternal cousins. Seven had hypoproteinemic edema and four of these suffered from hypocalcemic seizures. Chylous effusions were present in five; hypoproteinemia was present in 12 although five had no hypoalbuminemic edema. Six had lymphopenia which was related to the severity of the disease and was the last abnormality to disappear after clinical remission. Upper gastrointestinal tract series were performed in 13 children and had diagnostic supportive value in seven. Six children had two or more small intestinal biopsies done; all showed great variation from one examination to the other, ranging from a normal appearance to severe changes. Lymphatic block may occur at different sites: in the lamina propria only, generalized (lamina propria, submucosa, serosa, and mesentery), or conversely in the mesentery alone with minimal changes in the lamina propria. In three patients i.v. hyperalimentation was necessary. Specific treatment with a high-protein, low-fat diet with added medium-chain triglyceride is valuable. Surgical resection was of benefit in one patient and anastomosis of mesenteric to paraaortic lymph nodes in another. The data indicate that intestinal lymphangiectasia is more common than has been suspected and that the condition may vary widely in its manifestations and severity.

4545 BLEEDING MECKEL'S DIVERTICULUM DIAGNOSED BY ARTERIOGRAPHY AND RADIOISOTOPE IMAGING. *(E.)* Eisenberg, D.; Sherwood, C. E. (Highland Hosp., Rochester, N.Y.). *Am. J. Dig. Dis.* 20(6):573-576, 1975.

A 24-yr-old man with massive intestinal bleeding from a Meckel's diverticulum was diagnosed preoperatively by both abdominal arteriography and radioisotope imaging with technetium pertechnetate. A superior mesenteric arteriogram demonstrated pooling of contrast medium in the region of the distal ileum, to the right of the sacrum. The follow-up technetium imaging study showed accumulation of radioactivity corresponding to the angiographic pooling. At laparotomy, a large diverticulum of the ileum was found which contained ectopic gastric mucosa. The diverticulum and a short ileal segment were resected and an end-to-end anastomosis was performed. The surgical specimen revealed superficial erosion and ulceration of the ileal-bowel mucosa immediately adjacent to the diverticulum. When a bleeding Meckel's diverticulum is present, selective mesenteric artery arteriography may reveal either a localized area of vascularity in the right lower quadrant representing a richly vascular gastric tissue or puddling of contrast media near the site of active bleeding. Both these features existe in the present case The technetium pertechnetate ion is concentrated in functional ectopic tissue as well as in the stomach. Imaging is best performed with a gamma camera. False-negative findings in abdominal arteriography and technetium imaging may be caused by faulty technique or insufficient secretory gastric mucosa within the Meckel's diverticulum. False-positive findings have been reported

in association with intestinal hemangiomas, aneurysms, and intestinal ulceration. Both examinations appear to be promising adjuncts for the preoperative diagnosis of bleeding Meckel's diverticulum.

4546 CONGENITAL ATRESIA OF THE SMALL INTESTINE (FORMAL GENESIS, DIAGNOSIS AND THERAPY). *(Ger.)* Schickedanz, H.; Kleinteich, B. (Surg. Clin., Friedrich Schiller Univ., Jena, Germany). *Wiss. Z. Friedrich Schiller Univ. Jena Math. Naturwiss. Reihe* 23(3/4):625-636, 1974.

4547 FAMILIAL SYNDROME OF SHORT SMALL INTESTINE WITH INTESTINAL MALROTATION AND HYPERTROPHIC PYLORIC STENOSIS IN INFANTS. *(Fr.)* Royer, P.; Ricour, C.; Nihoul-Fekete, C.; Pellerin, D. (Hosp. Sick Child., Paris, France). *Arch. Fr. Pediatr.* 31(2/3):223-230, 1974.

4548 ROTATION ANOMALY AS CAUSE OF INTESTINAL OBSTRUCTION IN THE NEWBORN. *(Ger.)* May, Von D. A. (Child's Hosp. Med. Cent., Boston, Mass.). *Fortschr. Geb. Roentgenstr. Nulkearmed.* 121(2):179-185, 1974.

4549 INTESTINAL VOLVULUS. *(It.)* Mensorio, C.; Petrocchi, G.; Magnoni, E. (Inst. Clin. Pathol. Clin. Propedeut., Univ. Cagliari, Italy). *Chir. Ital.* 26(2):140-172, 1974.

4550 VASCULAR COMPRESSION OF THE DUODENUM. *(It.)* Dell'Anna, B. (Inst. Spec. Surg. Pathol. Clin. Propedeut., Univ. Parma, Italy). *Acta Chir. Ital.* 30(3):237-251, 1974.

4551 BENIGN TUMORS OF THE DUODENUM: LIPOMA. *(Fl.)* Kets, E.; Tanghe, W. (H. Hart Clin., Roeselare, Belgium). *Tijdschr. Gastroenterol.* 17 (2):71-86, 1974.

4552 RADIOGRAPHIC AND DUODENOSCOPIC VISUALIZATION OF POLYPOID CHANGES IN THE DUODENUM. *(Cz.)* Maratkia, Z.; Kocianova, J.; Pirk, F.; Kudrmann, J. (Bulovka Hosp., Prague, Czechoslovakia). *Cs. Gastroenterol. Vyz.* 29(1):7-12, 1975.

4553 PEUTZ-JEGHERS SYNDROME. *(E.)* Edelman, M. J.; Sternberg, S. S. (no affil.). *Clin. Bull.* 5(1):24-25, 1975.

4554 PRIMARY GRANULOCYTIC SARCOMA OF THE SMALL BOWEL. *(E.)* Brugo, E. A.; Larkin, E.; Molina-Escobar, J.; Costanzi, J. (Univ. Texas Med. Branch, Galveston). *Cancer* 35(5):1333-1340, 1975.

4555 INTESTINAL LEIOMYOSARCOMA STIMULATING MALIGNANT OVARIAN NEOPLASM. *(E.)* Ng, K. H.; Prathap, K. (Dep. Obstet. Gynecol. Pathol., Univ. Malaya, Kuala Lumpur, Malaysia). *Int. Surg.* 60(4): 237-238, 1975.

4556 MANIFEST INTESTINAL INVOLVEMENT DURING BONE-MARROW REMISSION IN A CASE OF ACUTE LYMPHOBLASTIC LEUKAEMIA. *(E.)* Aronson, A. S.; Garwicz, S.; Landberg, T.; Nelson, O.; Brun, A. (Univ. Hosp., Lund, Sweden). *Acta Pediatr. Scand.* 64(2):369-372, 1975.

4557 HODGKIN'S DISEASE OF THE DUODENUM PRESENTING WITH HAEMORRHAGE AND PERFORATION. *(E.)* Bardhan, K. D.; McArthur, P.; Rigby, C. (R. Hosp., Sheffield, England). *Postgrad. Med. J.* 51 (593):180-182, 1975.

4558 MALIGNANT TUMORS OF THE SMALL INTESTINE. *(It.)* Roviera, C.; Bologna, A.; Paganelli, F. (Inst. Gen. Clin., Surg., Univ. Turin, Italy). *Minerva Chir.* 29(22):1186-1195, 1974.

4559 PERFORATION OF A JEJUNAL NEUROFIBROMA. *(Rus.)* Usanov, A. P. (No. 28 Munic. Hosp., Gorky, USSR). *Vestn. Khir.* 114(2):130-131, 1975.

4560 THE DIAGNOSIS OF MYOGENOUS TUMORS OF THE SMALL INTESTINE. *(Rus.)* Mazanov, G. S.; Volchek, V. M.; Zolin, V. P. (Kalinin Med. Inst., USSR). *Klin. Khir.* (4):67, 1975.

4561 ABDOMINAL SYNDROME DUE TO SMALL INTESTINE METASTASES. *(Fr.)* Bernier, L.; Bertrand, M. B. (Hotel Dieu, Quebec, Canada). *Vie Med. Can. Fr.* 3(7):645-647, 1974.

4562 HEMOPERITONEUM DUE TO RUPTURE OF HEMANGIOPERICYTOMA OF THE SMALL BOWEL. *(It.)* Palagiano, V.; Liguori, L. (Clin. Gen. Surg. Surg. Ther., Univ. Bologna, Italy). *Acta Chir. Ital.* 30 (3):287-299, 1974.

4563 GIANT INTESTINAL LEIOMYOMA. *(Sp.)* De la Calle, F.; Senor Uria, C.; Riquelme, P.; Perez Sotelo, R.; Luna, A. (Virgen de Lluc Hosp., Palma de Mallorca, Spain). *Rev. Esp. Enferm. Apar. Dig.* 45(3):325-328, 1975.

4564 MULTIPLE LEIOMYOMAS OF THE SMALL INTESTINE MANIFESTED BY ACUTE ANEMIA. *(Fr.)* Aubert, P.; Lainee, R.; Lainee, J.; Fihey, M. A. (Paul Brousse Hosp., Villejuif, France). *Ann. Med. Interne* 126(4):279-283, 1975.

4565 TWO UNUSUAL CASES OF LEIOMYOMA OF THE DUODENOJEJUNAL JUNCTION. *(It.)* Bobbio, P.; Sacco, M. (Inst. Gen. Clin. Surg. Surg. Ther. I, Univ. Parma, Italy). *Acta Chir. Ital.* 30(3):279-285, 1974.

4566 PRIMARY RETICULUM CELL SARCOMAS OF THE SMALL INTESTINE. *(Ger.)* Werner, V. R. (Kaiser Franz Josef Hosp., Vienna, Austria). *Zentralbl. Allg. Pathol.* 118(4):390-392, 1974.

4567 INTRAPARIETAL HEMATOMA OF THE SMALL INTESTINE. *(Fr.)* Monnier, J. P.; Bigot, J. M.; Chermet, J.; Fragoas, M. (St. Antoine Univ. Hosp. Cent., Paris, France). *J. Padiol. Electrol.* 55(3):217-222, 1974.

4568 DUODENAL HEMATOMA. CRITICAL ANALYSIS OF RECENT TREATMENT TECHNICS. *(E.)* Janson, K. L.; Stockinger, F. (Bergstrom Air Force Base Hosp., Austin, Tex.). *Am. J. Surg.* 129(3):304-308, 1975.

4569 DUODENOJEJUNAL OBSTRUCTION CAUSED BY AN INTRAMURAL, POST-TRAUMATIC HEMATOMA. *(Fr.)* Boustany, F. N.; Atallah, P.; Khoury, P. (Hotel Dieu, Beirut, Lebanon). *J. Med. Liban* 27(5):531-542, 1974.

4570 GALLSTONE ILEUS. *(Rus.)* Mitov, E.; Manchev, I. (Plovdiv Med. Fac., Bulgaria). *Khirurgiia (Mosk.)* (3):88-89, 1974.

4571 SURGICAL PROCEDURES IN GALLSTONE ILEUS. *(Ger.)* Hantschmann, N.; Voigt, J.; Becher, R. (Cent. Surg. Med. I, Univ. Kiel, Germany). *Langenbecks Arch.* 338(1):55-65, 1975.

4572 A SIMPLE EFFECTIVE AID IN THE TREATMENT OF ILEUS. *(Dut.)* Coene, J. A.; Houthooft, D. H. C. (St. Elisabeth Hosp., Tilbury, Netherlands). *Ned. Tijdschr. Geneeskd.* 117(46):1727-1728, 1973.

4573 CALCULOUS OBSTRUCTION SYNDROME OF THE PYLORUS AND DUODENUM. *(It.)* Preziosi, M.; Imbesi, C. (S. Spirito Pio Inst., Rome, Italy). *Chir. Gastroenterol.* 8(3):335-341, 1974.

4574 A CASE OF EPITHELIOMA ON HETEROTOPIC MUCOSA FOUND WITHIN A PERFORATED MECKEL'S DIVERTICULUM. REVIEW OF THE LITERATURE. *(Fr.)* (Thomas, G.; Thomas, M.; Otteni, F.; Meyer, C. (no affil.). *Chirurgie* 100(11):791-794, 1974.

4575 MECKEL'S DIVERTICULUM AS A CAUSE OF ACUTE ABDOMINAL DISEASE. *(Rus.)* Valigura, Ia. S. (Lvov Med. Inst., USSR). *Klin. Khir.* (4):48-50, 1975.

4576 PERFORATION OF MECKEL'S DIVERTICULUM IN INFANCY. *(E.)* Canty, T.; Meguid, M. M.; Eraklis, A. J. (Child. Hosp. Med. Cent., Boston, Mass.). *J. Pediatr. Surg.* 10(2):189-194, 1975.

4577 RADIOLOGY OF MECKEL'S DIVERTICULUM. *(Fr.)* Bret, P.; Minh, V. T.; Boisson, M.; Saint-Cyr, M.; Deliot, J. F.; Burelle, H. (Eduoard Herriot Hosp., Lyon, France). *Ann. Radiol. (Paris)* 17(5):479-485, 1974.

4578 ONE CASE OF A BLEEDING LEIOMYOMA OF MECKEL'S DIVERTICULUM. *(It.)* Marin, A. W.; Nanni, G.; Pescatori, M. (Inst. Clin. Gen. Surg. Surg. Ther., Sacred Heart Cathol. Univ., Rome, Italy). *Chir. Patol. Sper.* 21(4):285-292, 1973.

4579 CURRENT PROBLEMS IN THE DIAGNOSIS AND TREATMENT OF INTESTINAL FISTULAS. *(Rus.)* Chkeidze, R. D. (Tbilisi State Med. Inst., USSR). *Soobshch. Akad. Nauk Gruz. SSR* 77(1):221-224, 1975.

4580 SURGICAL TECHNIQUES FOR EXTERNAL FISTULAS OF THE SMALL INTESTINE. *(Fr.)* Maillet, P. (no affil.). *J. Med. Strasbourg* 5(5):393-395, 1974.

4581 THE VALUE OF PARENTERAL HYPERALIMENTATION FOR THE THERAPY OF INTESTINAL FISTULAS. *(Fr.)* Leutenegger, A. F.; Goschke, H.; Schuman, L. (Dep. Surg., Univ. Basel, Switzerland). *J. Med. Strasbourg* 5(5):389-392, 1974.

4582 OUR EXPERIENCE WITH THE SURGICAL MANAGEMENT OF POSTOPERATIVE EXTERNAL FISTULAS OF THE SMALL BOWEL. *(Fr.)* Hollender, L. F.; Otteni, F.; Meyer, C.; Marrie, A. (no affil.). *J. Med. Strasbourg* 5(5):415-421, 1974.

4583 RUPTURE OF THE INTESTINE IN PATIENTS WITH HERNIA. *(E.)* O'Leary, J. P.; Macgregor, A. M. C. (Coll. Med., Univ. Florida, Gainesville). *South. Med. J.* 68(4):463-467, 1975.

4584 THERAPY OF INGUINAL HERNIAS IN CHILDHOOD. *(Ger.)* Kleinteich, B.; Schickedanz, H.; Glasel, E. (Surg. Clin., Friedrich Schiller Univ., Jena, Germany). *Wiss. Z. Friedrich Schiller Univ. Jena Math. Naturwiss. Reihe* 23(3/4):637-642, 1974.

4585 INTESTINAL INTUSSUSCEPTION. *(It.)* Mensorio, C.; Saracino, F.; Magnoni, E. (Inst. Surg. Pathol., Cagliari Univ., Italy). *Chir. Ital.* 26(3):230-245, 1974.

4586 RADIOLOGICAL SIGNS OF INTUSSUSCEPTION OF THE SMALL INTESTINE IN ADULTS: THREE CASES. *(Fr.)* Bret, P.; Piante, M.; Minh, V. T.; Boisson, M. (Eduoard Herriot Hosp., Lyon, France). *Ann. Radiol. (Paris)* 17(5):487-490, 1974.

4587 MULTIPLE SEGMENTAL DEMUSCULARIZATION IN THE TREATMENT OF SYNDROMES FOLLOWING EXTENSIVE INTESTINAL RESECTION. *(It.)* Perisi, P.; Arcerito, S.; Bacciu, P. P. (Inst. Clin. Gen. Surg. Surg. Ther., Univ. Sassari, Italy). *Chir. Patol. Sper.* 21(2):133-147, 1973.

4588 INVESTIGATION OF PATHOLOGICAL CHANGES IN THE INTESTINAL WALL. *(Ger.)* Bussmann, J. F.; Loewe, K. R.; Wemmer, U. (Surg. Clin., Univ. Heidelberg, Germany). *Bruns. Beitr. Klin.* 221(8): 605-610, 1974.

4589 PERFORATION AS A COMPLICATION OF JEJUNAL BIOPSY. *(Ger.)* Leinweber, B.; Schomerus, M. (Med. Clin., Justus Liebig Univ., Giesson, Germany). *Leber Magen Darm* 4(3):131-134, 1974.

4590 NON-SPECIFIC CHRONIC DUODENITIS: CONTEMPORARY DIAGNOSTIC POSSIBILITIES. *(Cs.)* Cheli, R.; Aste, H.; Ciancamerla, G. (San Marino Hosp., Genoa, Italy). *Cs. Gastroenterol. Vyz.* 28 (8):513-517, 1974.

4591 ENDOSCOPY OF THE UPPER AND LOWER DUODENUM. *(Cs.)* Benysek, L.; Kojecky, Z. (Intern. Med. Clin. II, Palacky Univ., Olomouc, Czechoslovakia). *Cs. Gastroenterol. Vyz.* 28(8):540-544, 1974.

4592 POSTOPERATIVE NECROTIZING ENTERITIS AFTER VAGOTOMY FOR DUODENAL BULB ULCER. *(Fr.)* Gautier-Benoit, G.; Pinchon-Marsy, S.; Vermesse, G.; Houcke, M. (no affil.). *Chirurgie* 100(11):777-780, 1974.

4593 METABOLIC INTESTINAL SURGERY. ITS COMPLICATIONS AND MANAGEMENT. *(E.)* Starkloff, G. B.; Donovan, J. F.; Ramach, K. R.; Wolfe, B. M. (St. Louis Univ. Hosp., Mo.). *Arch. Surg.* 110 (5):652-657, 1975.

4594 JEJUNO-ILEAL BYPASS AS TREATMENT OF INTRACTABLE OBESITY. *(E.)* Kosyak, E. P. (Hoopeston Comm. Mem. Hosp., Ill.). *Abdom. Surg.* 17(5):117-121, 1975.

4595 OBSERVATIONS ON THE HEALING OF WOUNDS OF THE INTESTINES. *(E.)* Ravitch, M. M. (Dep. Surg., Univ. Pittsburgh, Pa.). *Surgery* 77(5): 665-675, 1975.

4596 CYTOMETRIC ANALYSIS OF THE PARAVENTRICULAR NUCLEI IN RATS WITH OBSTRUCTION OF THE SMALL INTESTINE. *(Pol.)* Galasinska-Pomykol, I.; Stefanska-Sulik, E.; Chilimoniuk, M.; Oldak, B. (Inst. Biol. Morphol., Bialystok, Poland). *Endokrynol. Pol.* 25(5):361-368, 1974.

4597 DIGESTION AND ABSORPTION IN THE SMALL INTESTINE IN NORMAL SUBJECTS AND IN PATIENTS WITH CHRONIC ENTEROCOLITIS. *(Rus.)* Rybakova, G. S.; Zlatkina, A. R.; Ugolev, A. M. (M. F. Vladimirskii Moscow Reg. Res. Inst. Clin. Med., USSR). *Lab. Delo* (3):155-157, 1974.

4598 VILLOUS ADENOMAS AND MALIGNANT VILLOUS TUMOURS OF THE DUODENUM. CASE REPORT AND REVIEW OF LITERATURE. *(E.)* Dwyer, W. A.; O'Brien, R.; Maio, T. (St. Joseph's Hosp., Paterson, N.J.). *Conn. Med.* 39(5):286-290, 1975.

4599 DIVERTICULA OF MECKEL'S DIVERTICULUM. *(E.)* Kaneti, J.; Mares, A. J. (Soroka Med. Cent., Beersheva, Israel). *Am. J. Surg.* 129(5):597-598, 1975.

4600 SHORTENED SMALL BOWEL SYNDROME. MACKBY'S OPERATION. *(E.)* Camprodon, R.; Guerrero, J. A.; Salva, J. A.; Jornet, J. (Francisco Franco Munic. Sanit., Barcelona, Spain). *Am. J. Surg.* 129(5):585-586, 1975.

4601 CARCINOMA OF THE DUODENOJEJUNAL JUNCTION PRESENTING AS CHRONIC ANAEMIA. *(E.)* Martin, D. A. S.; Macarthur, E. (Australian Health Lab., Canberra). *Med. J. Aust.* 1(12):387-391, 1975.

See also, 3898, 4182, 4186, 4212, 4229, 4230, 4233, 4234, 4235, 4284, 4293, 4395, 4408, 4411, 4416, 4425, 4426, 4444, 4450, 4478, 4636, 4643, 4660, 4673, 4680, 4683, 4706, 4780, 4795, 4952, 5014, 5086, 5090, 5098, 5102, 5103, 5126, 5169, 5180, 5185, 5186, 5193.

SMALL INTESTINE

Malabsorption

4602 CONFIRMING PERSISTENCE OF GLUTEN INTOLERANCE IN CHILDREN DIAGNOSED AS HAVING COELIAC DISEASE IN INFANCY: USEFULNESS OF ONE-HOUR BLOOD XYLOSE TEST. *(E.)* Rolles, C. J.; Anderson, C. M.; McNeish, A. S. (Inst. Child Health, Univ. Birmingham, England). *Arch. Dis. Child.* 50(4): 259-263, 1975.

A p.o. gluten challenge (20 g/day) was given to 16 children diagnosed as having celiac disease when aged 3-11 months and who had been treated with a gluten-free diet for periods of 1 month to 5 yr. Xylose malabsorption was demonstrated in 15 children by a fall in the 1-hr blood xylose level within 1-28 days after gluten challenge. The fall tended

to be more rapid in children who had been on a gluten-free diet for less than 6 months and who had residual abnormalities in the jejunum. However, xylose malabsorption was rapidly produced in four children who had been on a gluten-free diet for 7-10 months, including two with a normal prechallenge jejunal histology. One child, with a persistently normal 1-hr blood xylose test after gluten challenge for 3 months, had normal absorption and normal jejunal histology after 18 months on a gluten-containing diet; she is considered not to have celiac disease. These results demonstrate that the 1-hr blood xylose test before and after gluten challenge is helpful in confirming the persistence of celiac disease diagnosed in infancy.

4603 MALABSORPTION SYNDROME WITH COW'S MILK INTOLERANCE: CLINICAL FINDINGS AND COURSE IN 54 CASES. *(E.)* Kuitunen, P.; Visakorpi, J. K.; Savilahti, E.; Pelkonen, P. (Child. Hosp., Univ. Helsinki, Finland). *Arch. Dis. Child.* 50(5): 351-356, 1975.

Clinical data are reported for 54 consecutive infants (25 boys and 29 girls) presenting with the malabsorption syndrome and cow's milk intolerance over a 10-yr period. All patients (mean age on admission, 15 weeks) had diarrhea, most had vomiting, and about 20% had atopic eczema and recurrent respiratory infections. Laboratory investigations revealed lactose malabsorption in about 1/2 the patients, raised serum IgA in 37 of 50, and precipitins to cow's milk in 32 of 49. Jejunal biopsy often showed severe mucosal damage of the type seen in celiac disease, and in about 1/2 the cases the mucosa was flat. The patients did well on human milk but reacted clinically to cow's milk challenge, either in a few hrs (28 cases) or gradually during 3-4 weeks (26); in some patients an initial rapid and a later slow reaction was discernible. Clinical symptoms of cow's milk intolerance disappeared at about age 1 yr, when 81% had normal fecal fat but only 29% a normal proximal jejunal mucosa. Positive reactions to cow's milk protein fractions were most frequent with β-lactoglobulin and casein. Sensitivity to cow's milk was also associated with intolerance to other food proteins such as soya and wheat. Of the 42 patients followed for 2 yr on a gluten-containing diet, 37 have a normal or nearly normal jejunal mucosa and 5 (12%) have subtotal villous atrophy indicative of celiac disease. It is concluded that the malabsorption syndrome with cow's milk intolerance is a clear-cut clinical entity. However, the symptomatology, results of laboratory tests, and jejunal biopsy findings closely resemble those of other entities where damage to the intestinal mucosa causes a malabsorption syndrome. Follow-up studies indicate that the disease is transient, but in about 10% of patients the primary disorder is celiac disease.

4604 ENTEROTOXIGENIC INTESTINAL BACTERIA IN TROPICAL SPRUE. II. EFFECT OF THE BACTERIA AND THEIR ENTEROTOXINS ON INTESTINAL STRUCTURE. *(E.)* Klipstein, F. A.; Schenk, E. A. (Trop. Malabs. Unit, Univ. Rochester, New York). *Gastroenterology* 68(4):642-655, 1975.

Randomly selected strains of the predominant coliform bacteria (3 serotypes of *Klebsiella pneumoniae* and a strain of *Enterobacter cloacae)* isolated from each of 4 Puerto Ricans with tropical sprue were examined for their effect on intestinal structure in the ileal loop model of 34 rabbits. The viable bacteria did not evoke a fluid response, invade the mucosa, or cause changes in villus architecture; but they did result in the formation of a luminal exudate consisting of mucin, necrotic epithelial cells, bacteria, and epithelial cell enzymes. Cell-free broth filtrates of bacterial growth of all 4 strains produced an increasingly positive fluid response from 2-18 hr and progressively more severe structural changes consisting of shortening and blunting of the villi, infiltration predominantly by mononuclear cells, and submucosal congestion. Equally hypermolar control preparations of broth-free medium without bacterial growth and cell-free broth filtrates of a nontoxigenic strain of *Escherichia coli* failed to induce these abnormalities. These observations indicate that the coliform bacteria tested elaborate a toxic product, presumably an enterotoxin, which produces structural and functional changes of the intestinal mucosa resembling those present in tropical sprue.

4605 ILEAL FUNCTION IN PATIENTS WITH UNTREATED ADULT COELIAC DISEASE. *(E.)* Silk, D. B. A.; Kumar, P. J.; Webb, J. P. W.; Lane, A. E.; Clark, M. L.; Dawson, A. M. (St. Bartholomew's Hosp., London, England). *Gut* 16(4):261-267, 1975.

Jejunal and ileal absorption of glucose, water, and electrolytes in 8 patients (mean age, 38 yr) with untreated adult celiac disease was investigated with a double-lumen perfusion technique. Correct positioning of the tube was confirmed by measuring the differential jejunal and ileal handling of bicarbonate. The 8 patients and 8 controls (mean age, 40 yr) were perfused with an isotonic electrolyte solution containing 50 mM glucose and 25 mM bicarbonate. Compared with the controls, the celiac patients had significantly impaired jejunal absorption of glucose, water, sodium, and chloride. In contrast, the celiac patients had normal ileal glucose and water absorption and significantly increased ileal sodium and chloride absorption compared with the controls. Evidence for ileal adaptation was found in 3 patients who had absorptive values outside 2 standard deviations of the normal mean. These patients had greater ileal surface cell heights than the other celiac patients and tended to have taller villi than the remaining celiacs. This study indicates that the distal small intestine in celiac disease has the ability to adapt to the damage and loss of absorptive capacity in the proximal small intestine. The enhanced ileal function in celiac disease may be due to distal ileal mucosal cell hyperplasia ocurring in response to the jejunal mucosal lesion.

4606 SMALL INTESTINAL BIOPSY IN COW'S MILK PROTEIN ALLERGY IN INFANCY. *(E.)* Fontaine, J. L.; Navarro, J. (Trousseau Hosp., Paris, France). *Arch. Dis. Child.* 50(5):357-362, 1975.

Intestinal biopsies were performed in 31 infants (19 girls and 12 boys, aged 3-30 months) with cow's milk protein intolerance. Mucosal damage was observed in 30 cases, including 4 infants biopsied while on a milk-free diet. Partial villous atrophy was present in 25 cases, subtotal villous atrophy in 1, and slight histological lesions in 4. Of the 25 cases of partial villous atrophy, 5 were Grade I; 12, Grade II; and 8, Grade III. Complete mucosal recovery occurred in 4 children after 3-13 months on a milk-free diet. It is concluded that the mucosal damage seen in cow's milk protein allergy is induced by cow's milk, though the mechanism remains unknown.

4607 CELL-MEDIATED IMMUNITY TO GLIADIN WITHIN THE SMALL-INTESTINAL MUCOSA IN COELIAC DISEASE. *(E.)* Ferguson, A.; MacDonald, T. T.; McClure, J. P.; Holden, R. J. (West. Infirm., Glasgow, Scotland). *Lancet* (7912):895-897, 1975.

In an attempt to demonstrate local cell-mediated immunity to gliadin in patients with celiac disease, fragments of jejunal biopsy specimens were cultured in the presence or absence of α-gliadin and the culture medium assayed for its capacity to inhibit migration of normal human peripheral blood leukocytes (i.e., for a migration-inhibition factor MIF). No MIF activity was detected in the culture medium when biopsy specimens from 15 patients with celiac disease or from 29 controls were cultured without added gliadin. The addition of α-gliadin (0.5 mg/ml) to the culture system did not result in MIF secretion by nonceliac mucosa or by the mucosa from 2 patients with celiac disease treated with a gluten-free diet. However, when biopsy specimens from 6 patients with untreated celiac disease were cultured with α-gliadin, they secreted a factor into the culture medium which significantly inhibited the migration of normal human leukocytes (>30% inhibition in 5 of the 6 cases). These findings suggest that there is a population of lymphocytes which are sensitized to gliadin in the intestinal mucosa of patients with untreated celiac disease. They support the theory that a local cell-mediated immune reaction may be the cause of villous atrophy and crypt hyperplasia in celiac disease.

4608 CHARACTERISTICS OF CARBOHYDRATE ABSORPTION AND METABOLISM IN PREMATURE AND NEWBORN INFANTS. *(Ger.)* Beyreiss, K.; Rautenbach, M.; Willgerodt, H.; Schone, S.; Al-Rebate, I. (Pediatr. Clin., Karl Marx Univ., Leipzig, Germany). *Wiss. Z. Friedrich Schiller Univ. Jena Math. Naturwiss. Reihe* 21 (4):683-693, 1972.

Parameters of the absorption and conversion of the fructose and galactose were studied using blood level kinetic procedures in premature, newborn, nursing infants, and healthy children and adults. Total clearance, absorption rate (Dost), and the concentration of the hexoses in blood, urine and stool were measured by specific methods. Characteristics of active monosaccharide absorption with adequate transport capacity in neonates was demonstrated in the first days of life. The low absorption rates of fructose in all age groups were caused in part by transformation in the small intestinal mucosa. For galactose also, a conversion upon passage through the intestinal mucosa was assumed with neonates and nursing infants in contrast to adults. The conversion rate of fructose was depressed in the neonatal period independent of maturity and attained the parameters of later infant age in the third week of life. In contrast, the optimal conversion range and the maximum conversion capacity for galactose with newborns was considerably greater than with adults. True premature infants showed hindered conversion of galactose only in the first days of life as compared to matured neonates. In the neonatal period, galactose raises the glucose concentration in the blood independent of the entrance location; fructose, however, raises blood glucose concentration only when given p.o.

4609 ABNORMALITIES OF SERUM CHOLECYSTOKININ AND GALLBLADDER EMPTYING IN CELIAC DISEASE. *(E.)* Low-Beer, T. S.; Harvey, R. F.; Davies, E. R.; Read, A. E. (R. Infirm., Bristol, England). *N. Engl. J. Med.* 292(18):961-963, 1975.

The relation between gallbladder contraction and serum cholecystokinin levels after a fatty meal was studied in 10 patients (7 men and 3 women, average age 58 yr) with adult celiac disease and 10 controls with no history of upper-intestinal tract disease. After an overnight fast, each subject drank 60 ml of a flavored emulsion of 33% peanut oil. Serum cholecystokinin levels, at various intervals after consumption of the emulsion, were measured by a radioimmunoassay technique; gallbladder emptying was monitored continuously *via* isotope cholecystography. In 9 of the patients, the fasting serum cholecystokinin levels were significantly raised over control values. However, the peak serum cholecystokinin levels and the increments in serum cholecystokinin after fat intake were lower in the celiac disease patients. Emptying of the gallbladder was less complete in the patients than in the controls and the onset of gallbladder emptying was significantly delayed in the former. The elevated fasting serum cholecystokinin levels in the celiac disease patients may have resulted from increased secretion of the hormone by the enterochomaffin-like cells in the small-bowel mucosa; this might result from a disturbance in some regulatory mechanism for hormone secretion. Available evidence suggests that the gallbladder in celiac disease is less responsive than normal to the action of endogenous cholecystokinin. The pathogenesis of the diminished pancreatic exocrine response which has been noted in celiac disease is probably similar to that of the diminished gallbladder contraction. Resistance to the action of cholecystokinin in both cases may in some way be related to the presence of constantly high levels of this hormone in the blood.

4610 ULTRASTRUCTURAL AND HISTOCHEMICAL STUDY OF THE BACTERIAL DESTRUCTION IN TWO CASES OF WHIPPLE'S DISEASE. *(Fr.)* Chamlian, A.; Casanova, P.; Cougard, A.; Mathieu, B. (Fac. Med., Marseille, France). *Ann. Anat. Pathol. (Paris)* 19(2):221-230, 1974.

The destruction of bacteria in two men aged 40 and 45 with Whipple's disease was studied by examination of specimens of jejunal mucosa obtained by peroral biopsy from the duodeno-jejunal flexure, both before and after a 7-month course of treatment which established remission. In one case the treatment was 1 g/day erythromycin, and in the other case it was 0.2 g/day doxycyclin. In the untreated cases, bacteria were concentrated in the mucous chorion of the intestinal villi and may accumulate under the basal epithelial basal membrane at the apex of the villi. Bacteria are often located around fatty particles and are often vacuolized. When they penetrate the villous chorion, they are attacked and digested by macrophages. At the inferior part of the mucosa, between the glandular cul-de-sacs and the muscularis mucosae, the macrophages seem to be less active than at the top of the villi, as they do not contain bacterial bodies but rather granular or filamentous material. During remission, bacteria are no longer visible, and the residual macrophages contain lamellate or granular material. In the untreated disease, the macrophages at the top of the villi are strongly colored by para-aminosalicylic acid which indicates the presence of neutral mucopolysaccharides. They have no affinity for colloidal iron, whereas the macrophages at the inferior part of the mucosa have the opposite affinities. During remission, the few remaining macrophages at the top of the villi react with both para-aminosalicylic acid and colloidal iron, while the more numerous deeper macrophages contain occasional particles reactive with para-aminosalicylic acid and many voluminous vacuoles with a moderate reaction to colloidal iron. The para-aminosalicylic acid undoubtedly is made to react by the mucopeptides contained in the bacterial wall. Acid phosphatase activity is great in the superficial macrophages and moderate in the deeper macrophages in the untreated disease. Apparently, macrophages at the top of the villi ingest material and migrate deeper into the mucosa as they digest it.

4611 CLINICAL, IMMUNOLOGICAL AND ELECTRON MICROSCOPE STUDIES OF WHIPPLE'S DISEASE. *(Ger.)* Buchholz, K.; Maintz, J.; Otto, H. F. (Hamberg-Heidberg Hosp., Germany). *Klin. Wochenschr.* 52(14):672-677, 1974.

An unusual course of Whipple's disease is reported. The patient (male, 57 yr) had a stomach resection for bleeding ulcer about 1/2 year prior to admission and was in generally poor health; malabsorption was suspected. Small intestinal biopsy was performed and Whipple's disease was confirmed on the basis of light and electron microscope evidence. Treatment with tetracycline (Vibramycin, 100 mg/day) resulted in rapid disappearance of clinical symptoms, weight gain, and return to normal levels of blood chemical data. A persistent clinical remission was noted 1 yr later with continued antibiotic therapy. As early as 5 months after institution of therapy multiple control biopsies showed a strong and progressive collagen formation of the stratum propium mucosae, especially in the pericellular region and near the epithelial basal membrane. The number of lymphocytes, plasma cells and interepithelial lymphoid round cells increased considerably. Immunologically, the serum complement (C_3-fraction) was decreased. Immunohistologically, subepithelial and interepithelial cells were found after incubation with the patient's serum showing a cellular-cytoplasmic fluorescence with anti-complement. These findings indicate a hyper-reactive mechanism possibly leading to induction of a cellular immune process.

4612 HISTOCHEMICAL STUDY OF ENTEROKINASE IN DUODENAL AND JEJUNAL BIOPSIES FROM CHILDREN WITH MALABSORPTION SYNDROME. *(Cz.)* Lojda, Z.; Jodl, J. (Fac. Gen. Med., Charles Univ., Prague, Czechoslovakia). *Cesk. Gastroenterol. Vyz.* 28(8):532-539, 1974.

Since histological studies of unaffected parts of the duodenum and jejunum, removed from two patients at surgery, demonstrated that enterokinase activity is much higher in the duodenum than in the jejunum, duodenal biopsies were taken from children with a variety of malabsorption syndromes. Using a method described previously, the enterokinase activity in duodenal biopsies taken from the mid-duodenum in patients without gastrointestinal symptoms reached a maximum in the enterocytes after 2 hr of incubation and was so high on the top and along the sides of the villi that a diffuse reaction product was seen as far as the lamina propria. After this incubation period, the cytoplasm of Brunner's glands was stained only nonspecifically by the diazo reaction and the enterokinase activity in the epithelium of the crypts was very low. Enterokinase activity in duodenal biopsies from three children with pancreatogenic malabsorption was similar to that found in five children with no evidence of gastrointestinal disease. Normal enterokinase activities were also found in duodenal biopsies from two children with lactase deficiency, as diagnosed by jejunal biopsies, and from five of nine children with diarrhea of unknown etiology; enterokinase activities were decreased in the remaining four cases. Only moderate irregularities in mucosal relief and moderate inflammatory changes were observed in these cases of diarrhea. Duodenal enterokinase activities were normal in four and decreased in eight patients with cow's milk intolerance and normal lactase activities. No enterokinase at all was present in the duodenum of the remaining four patients, even when biopsies were repeated. Duodenal enterokinase activities were normal in 12 and decreased in 12 children with manifest celiac disease. Of seven patients with celiac disease in remission, five exhibited normal duodenal enterokinase activity and two decreased activity. In four cases (one patient each with disaccharide deficiency, cow's milk intolerance, manifest celiac disease and celiac disease in remission), biopsies from the distal duodenum had no evidence of enterokinase activity.

4613 THE CURRENT TENDENCY TO RECONSIDER THE CLASSIFICATION OF MALABSORPTION SYNDROMES: GLUTEN-INDUCED ENTEROPATHY. *(Rum.)* Iancu, A. Th.; Iancu, A. V. (Med. Pharm. Inst., Cluj, Rumania). *Rev. Med. Chir.* 78(2):283-288, 1974.

4614 PROTEIN-LOSING ENTEROPATHY IN A SIX-MONTH-OLD INFANT. *(Rus.)* Loseva, A. G.; Novozhilova, Z. I. (I. I. Pavlov Med. Inst., Leningrad). *Pediatriia* (3):83-84, 1975.

4615 ENZYMES AND ABSORPTION IN THE SMALL INTESTINE IN ELDERLY SUBJECTS. *(Rus.)* Valenkevich, L. N. (Leningrad Inst. Pediatr. Med., USSR). *Klin. Med. (Mosk.)* 52(7):89-93, 1974.

4616 DIAGNOSTIC AND THERAPEUTIC ASPECTS OF DISORDERS IN ABSORPTION AND DIGESTION IN ELDERLY SUBJECTS. *(Ger.)* Dietze, F.; Laue, R.; Schultz, H. J.; Kalhe, I. (Med. Clin., Humboldt Univ., Berlin, Germany). *Z. Alternsforsch.* 28(2):125-135, 1974.

4617 CLINICAL ASPECTS AND TREATMENT OF DISORDERS IN BILE ACID METABOLISM OCCURRING IN DISEASES OF THE SMALL INTESTINE. *(Ger.)* Fromm, H. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:421-427, 1974.

4618 SERUM LEVELS OF VITAMIN A (RETINOL) AND RETINOL-BINDING-PROTEIN (RBP) IN MALABSORPTION, DIGESTIVE DISEASES, AND CIRRHOSIS. *(Ger.)* Prellwitz, W.; Olbermann, M.; Kapp, S.; Muller, D.; Groth, U. (Med. Clin. II, Univ. Mainz, Germany). *Klin. Wochenschr.* 52(19):921-925, 1974.

4619 CALCIUM ABSORPTION IN OBESE SUBJECTS DURING FASTING. *(Cz.)* Kocian, J.; Rath, R. (Inst. Postgrad. Med. Pharm., Prague, Czechoslovakia). *Cesk. Gastroenterol. Vyz.* 28(7):454-460, 1974.

4620 PERORAL BIOPSIES OF THE INTESTINAL MUCOSA. COMPARITIVE STUDIES ON THE HISTOLOGICAL AND DISSECTING MICROSCOPE FINDINGS. *(Ger.)* Schmidt, K.; Scholl, A. (Pediatr. Clin., Univ. Tubingen, Germany). *Z. Kinderheilk.* 191(1):37-45, 1975.

4621 COW'S MILK PROTEIN INTOLERANCE. TRANSIENT FOOD INTOLERANCE OF INFANCY. *(E.)* Walker-Smith, J. (R. Hosp. St. Bartholomew, London, England). *Arch. Dis. Child.* 50(5):347-350, 1975.

4622 THE MORPHOLOGY OF THE SMALL INTESTINE IN DISORDERS OF MALABSORPTION. *(E.)* Duffell, D. R. (Northwest Commun. Hosp., Chicago, Ill.). *Proc. Inst. Med. Chic.* 30(5):170, 1974.

4623 JEJUNAL HYDROLYSIS AND UPTAKE OF CONJUGATED FOLATE IN TROPICAL SPRUE. *(E.)* Halsted, C. H.; Reisenauer, A. M.; Corcino, J. J. (Dep. Med., Univ. California, Davis). *Clin. Res.* 23(3):457A, 1975.

4624 DOMESTIC SPRUE - A DISEASE ENTITIY FREQUENTLY NOT RECOGNIZED. *(Ger.)* Herms, G.; Kramer, G. (Magdeburg-Altstadt Dist. Hosp., Germany). *Z. Gesamte. Inn. Med.* 30(5):178-183, 1975.

4625 WHIPPLE'S DISEASE. CASE REPORT. *(Fr.)* DeWulf, E.; Vielle, G.; Hongque, M. P.; Arianoff, A. A. (St. Elisabeth Clin., Brussels, Belgium). *Acta Gastroenterol. Belg.* 37(4/5):207-224, 1974.

4626 INTESTINAL LIPODYSTROPHY (WHIPPLE'S DISEASE). REPORT OF A CASE WITH NEARLY FATAL OUTCOME. *(Ger.)* Gobel, D. (Richt Hosp., Karlsruhe, Germany). *Leber Magen Darm* 4(3):117-122, 1974.

4627 INCIDENCE OF CELIAC DISEASE IN AUSTRIAN CHILDREN. *(Ger.)* Rossipal, E. (Pediatr. Clin., Univ. Graz, Austria). *Z. Kinderheilkd.* 119 (2):143-149, 1975.

4628 MORPHOLOGICAL AND BIOCHEMICAL CHANGES IN THE SMALL INTESTINAL MUCOSA IN BLIND-LOOP SYNDROME. *(Ger.)* Bloch, R.; Menge, H.; Lorenz-Meyer, H.; Riecken, E. O. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:853-856, 1973.

4629 CHEMISTRY OF WHEAT PROTEINS AND THE NATURE OF THE DAMAGING SUBSTANCES. *(E.)* Evans, D. J.; Patey, A. L. (no affil.). *Clin. Gastroenterol.* 3(1):199-211, 1974.

4630 BARIUM STUDIES. *(E.)* Burrows, F. G. O.; Toye, D. K. M. (no affil.). *Clin. Gastroenterol.* 3(1):91-107, 1974.

4631 CLINICAL AND MORPHOLOGIC RESPONSE TO GLUTEN WITHDRAWAL. *(E.)* Steward, J. S. (no affil.). *Clin. Gastroenterol.* 3(1):109-126, 1974.

4632 THE DISORDER IN CHILDHOOD. *(E.)* McNeish, A. S.; Anderson, C. M. (no affil.). *Clin. Gastroenterol.* 3(1):127-144, 1974.

4633 ELECTRON MICROSCOPY OF JEJUNAL MUCOSA. *(E.)* Shiner, M. (no affil.). *Clin. Gastroenterol.* 3(1):33-53, 1974.

4634 ANAEMIA IN ADULT COELIAC DISEASE. *(E.)* Hoffbrand, A. V. (no affil.). *Clin. Gastroenterol.* 3(1):71-89, 1974.

4635 MORPHOLOGY AND CELL KINETICS OF THE JEJUNAL MUCOSA IN UNTREATED PATIENTS. *(E.)* Watson, A. J.; Wright, N. A. (no affil.). *Clin. Gastroenterol.* 3(1):11-31, 1974.

4636 MALIGNANCY. *(E.)* Stokes, P. L.; Holmes, G. K. T. (no affil.). *Clin. Gastrointerol.* 3(1):159-170, 1974.

4637 ACID MICROCLIMATE IN INTESTINAL ABSORPTION. *(E.)* Blair, J. A.; Matty, A. J. (no affil.). *Clin. Gastroenterol.* 3(1):183-197, 1974.

4638 THE SMALL INTESTINE AT AUTOPSY. *(E.)* Thompson, H. (no affil.). *Clin. Gastroenterol.* 3(1):171-181, 1974.

4639 DERMATITIS HERPETIFORMIS. *(E.)* Fry, L.; Seah, P. P.; Hoffbrand, A. V. (no affil.). *Clin. Gastroenterol.* 3(1):145-157, 1974.

4640 COELIAC DISEASE: IMMUNOLOGY. *(E.)* Asquith, P. (no affil.). *Clin. Gastroenterol.* 3(1):213-234, 1974.

4641 DRUG-INDUCED MALABSORPTION. *(E.)* Longstreth, G. F.; Newcomer, A. D. (Mayo Grad. Sch. Med., Rochester, Minn.). *Mayo Clin. Proc.* 50(5):284-293, 1975.

4642 COELIAC DISEASE IN CHILDHOOD. *(E.)* Wharton, B. A. (Queen Elizabeth Med. Cent., Birmingham, England). *Br. J. Hosp. Med.* 12(4):452-466, 1974.

4643 ADULT CELIAC DISEASE COMPLICATED BY INTESTINAL RETICULUM CELL SARCOMA WITH HIGH SERUM IgA LEVEL. *(E.)* Mussche, M.; Thienpont, L. (Acad. Hosp., State Univ. Ghent, Belgium). *Acta Clin. Belg.* 29(6):388-393, 1974.

4644 LACTOSE AND MILK INTOLERANCE: CLINICAL IMPLICATIONS. *(E.)* Bayless, T. M.; Rothfeld, B.; Massa, C.; Wise, L.; Paige, D.; Bedine, M. S. (Perry Point VA Hosp., Md.). *New Engl. J. Med.* 292(22):1156-1159, 1975.

See also, 4190, 4262, 4407, 5045, 5047, 5049, 5113.

LARGE INTESTINE

4645 NEW TECHNIQUE FOR THE STUDY OF DEFECATION BY MANOMETRY: ITS APPLICATION IN THE DIFFERENTIAL DIAGNOSIS OF MEGARECTUM. *(Fr.)* Hata, Y.; Duhamel, B.; Labarraque, J. P. (Saint-Denis Hosp. Cent., France). *Ann. Chir. Infant.* 15(2):77-86, 1974.

A simple manometric technique was used to visualize the rectoanal inhibitory reflex in 4 normal 3-yr-old children. A balloon-tipped probe was inserted in the barium-filled rectum until the ballon reached the upper portion of the internal sphincter. The balloon was then rapidly inflated while a series of profile X-rays was taken of the seated child. A minimum balloon volume of $10cm^3$ was necessary to stimulate opening of the anal canal, which was widest 3-4 sec after stimulation and closed again after 7 sec. A 4-yr-old girl with hypoganglionic megarectum needed a balloon volume of $50cm^3$ to open the canal, which then took 10 sec to close. This technique has shown that the internal and external anal sphincters react together, and not sequentially, on dilatation of the rectum. When perfected, it should prove a valuable aid in the differential diagnosis of different types of megarectum.

4646 CEA AS A MONITOR OF GASTROINTESTINAL MALIGNANCY. *(E.)* Holyoke, E. D.; Ming Chu, T.; Murphy, G. P. (Roswell Park Mem. Inst., Buffalo, N. Y.). *Cancer* 35(3):830-836, 1975.

Patients (94) with carcinoma of the colon were studied to determine the reliability of carcinoembryonic antigen (CEA) as a monitor of the malignancy. Determinations of plasma CEA levels were made over a 3-yr period using the Hansen assay, and simultaneous measurements of alkaline phosphatase were made. The CEA levels in 719 of 912 determinations accurately reflected the presence or absence of tumor. The assay detected 47 of 52 clinically clear-cut progressions; the CEA levels rose for 6-14 months in advance of clinical progression. False positive results were encountered in 10% of the patients. CEA levels responded to chemotherapy in close correlation with observed clinical course in the patients with metastatic disease. Initial pretherapy CEA values proved to be good prognostic indicators of disease course following complete resection; an initial CEA value less than 2.5 ng/ml plasma had recurrence of 1 in 120, a CEA greater than 7.0 ng/ml had recurrence of 7 in 9. CEA measurement was found to be as much as twice as sensitive as the alkaline phosphatase test, particularly for metastatic tumor in the liver. The results indicate that CEA can be used as a monitor of patients with colon carcinoma providing its limitations are understood.

4647 METASTATIC CARCINOMA SIMULATING INFLAMMATORY COLITIS. *(E.)* Meyers, M. A.; Oliphant, M.; Teixidor, H.; Weiser, P. (New York Hosp.-

Cornell Univ. Med. Cent., N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(1):74-83, 1975.

Cases (12) of metastatic carcinoma which were initially considered as ulcerative or granulomatous colitis clinically and roentgenologically were studied to establish distinguishing features of the malignancies. Characteristic roentgen findings included mucosal thickening, nodular masses, multiple and eccentric strictures, asymmetric involvement, pseudosacculations, spiculations of contour, and frequent associated terminal ileal involvement. Discrete changes and the distribution of colonic involvement distinguished metastatic disease from inflammatory colitis; the specific features were dependent upon pathways of spread, growth characteristics, and local tissue response. Where differentiation is difficult, deep biopsies, histologic documentation or other roentgenographic studies are suggested. Recognition of the characteristic roentgenologic changes is of great clinical importance and leads to a search for the primary neoplasm or establishes the diagnosis of the disease.

4648 MESOCOLIC LYMPH NODE HISTOLOGY IS AN IMPORTANT PROGNOSTIC INDICATOR FOR PATIENTS WITH CARCINOMA OF THE SIGMOID COLON: AN IMMUNOMORPHOLOGIC STUDY. *(E.)* Patt, D. J.; Brynes, R. K.; Vardiman, J. W.; Coppleson, L. W. (Univ. Chicago Hosp. Clin., Ill.). *Cancer* 35(5):1388-1397, 1975.

Histologic changes in human lymph nodes draining adenocarcinomas of the sigmoid colon were evaluated and the prognostic significance of the observed changes were assessed. Cases of adenocarcinoma of the sigmoid colon (36) treated between 1965 and 1968 were studied. Lymph nodes were examined for germinal center activity, sinus histiocytosis, and paracortical activity. Patients whose lymph nodes showed morphological evidence of cell-mediated immunity, either by an increased number of paracortical immunoblasts or sinus histiocytosis, survived significantly longer than those whose lymph nodes showed no such changes. Patients whose lymph nodes showed simultaneous paracortical activity and sinus histiocytosis had the best survival of all. Of that group, 11 of 13 lived 5 yr or more after surgery without signs of recurrent tumor. Germinal center activity was not a significant factor in prognosis. Favorable lymph node histology did not significantly correlate with the modified Duke's classification. The observations support the hypothesis that the immunoblasts observed within the paracortical region of draining lymph nodes are a morphological manifestation of an antitumor T-cell-mediated immune response, and those patients whose lymph nodes have a morphological pattern compatible with a cell-mediated immune response have the best prognosis.

4649 PAPILLARY ADENOMAS OF THE COLON AND RECTUM: A TWELVE-YEAR REVIEW. *(E.)* Jahadi, M. R.; Bailey, W. (Baylor Univ. Med. Cent., Dallas, Tex.). *Dis. Colon Rectum* 18(3):249-253, 1975.

A retrospective study was made to review 12-yr experience with papillary adenomas of the colon and rectum and to evaluate methods of management. Between 1960 and 1972, 185 patients (101 women, 84 men) with papillary adenomas of the colon and rectum were treated; the average age was 63 yr. Of these patients, 40% had rectal bleeding; 71% of the lesions occurred in the rectum and rectosigmoid. The lesions were malignant in 52% of cases; 9% of the patients died with metastatic carcinoma. The 5-yr survival rate was 75%. The results of the study indicate that a conservative approach to the surgical management of papillary adenoma of the colon and rectum should be encouraged, unless the tumor contains or is suspected to contain invasive carcinoma.

4650 APPENDICITIS. A CRITICAL REVIEW OF DIAGNOSIS AND TREATMENT IN 1,000 CASES. *(E.)* Lewis, F. R.; Holcroft, J. W.; Boey, J.; Dunphy, J. E. (San Francisco Gen. Hosp., Calif.). *Arch. Surg.* 110(5):677-684, 1975.

A retrospective analysis was made of 1,000 cases of appendicitis seen from 1963 to 1973 to provide demographic data, to elucidate reliable symptoms, signs, and laboratory studies, and to determine factors affecting morbidity and mortality of appendicitis. The overall negative appendectomy rate was 20%, but it exceeded 40% in women aged 20-40 yr. Nonsurgical lesions were the cause of 2/3 of the negative appendectomies; mesenteric adenitis, gastroenteritis, and abdominal pain of unknown cause accounted for 1/3 of the errors in females and 2/3 in males. Those diseases were best distinguished from appendicitis on the basis of temperature and white blood cell count. The remainder of errors in females were due to pelvic inflammatory disease or other gynecologic problems and were best distinguished from appendicitis on the basis of history and physical findings. The rate of perforation was 21% and the incidence of wound infection was 8.5%. Use of antibiotics did not affect wound infection rate, but they are clearly indicated in perforated appendicitis. Morbidity from appendicitis was primarily due to infection; it was noted that vertical incisions had a significantly higher incidence of infection than right lower quadrant oblique incisions. The authors express the need for improved accuracy in early diagnosis of appendicitis to reduce the rate of unnecessary appendectomy.

4651 ANORECTAL AND COLONIC MANOMETRY: NORMAL AND PATHOLOGICAL MOTOR ACTIVITY. *(Por.)* Habr-Gama, A.; Haberkorn, S.; Gama-Rodrigues, J. J.; Raia, A.; Bettarello, A. (Fac. Med., Univ. Sao Paulo, Brazil). *Arq. Gastroenterol. Sao Paulo* 11(4):201-216, 1974.

Analysis was made of 268 tracings of motor activity in the left colon and rectum (88 cases) or in the anorectal region (180 cases). Colonic manometry was performed on 15 normal controls, five patients with constipation, five with diarrhea, 48 with megacolon due to trypanosomiasis, and 15 with diverticula of the colon. Anorectal tracings were obtained from 15 normal children, 95 with constipation, 30 with congenital megacolon, 20 with malformations of the

lumbosacral spine, and 20 patients with megacolon due to trypanosomiasis. Colonic manometry gave the best information on the physiopathology in many of these conditions and served as an aid in selecting methods of treatment. In chronic constipation, colonic motility was increased, suggesting that the use of antispasmodics would facilitate passage of stools. In diarrhea, segmental colonic contractions were decreased, suggesting that anticholinergics, rather than antispasmodics, should be employed. Colonic manometry also demonstrated that diverticula cause increased segmental pressures, primarily in the sigmoid. In uncomplicated forms of diverticulosis and at its onset a high-residue diet facilitates the passage of stools and might prevent the formation of segmental chambers which lead to diverticula. If this conservative therapy fails, sigmoidomyotomy might decrease intraluminal pressures. Megacolon due to trypanosomiasis was accompanied by dyskinesia and absence of the inhibitory reflex at the internal anal sphincter after rectal distention. On the basis of these findings, it is concluded that surgery only improves symptoms by removing the enlarged colon and correcting fecal stasis without having any effect on underlying physiological changes. In children with congenital megacolon, anorectal manometry helps to establish a differential diagnosis between constipation and congenital megacolon, even when the aganglionic segment is very short and a barium enema gives doubtful results. In children with various spinal malformations (sacral agenesis, meningocele, myelomeningocele, and occult spina bifida), anorectal manometry demonstrated whether pressures at the level of the internal anal sphincter were normal and whether this muscle could be used in training these children to develop a normal rhythm of evacuation.

4652 CLINICAL PATTERNS OF ABDOMINAL TUBERCULOSIS. *(E.)* Das, P.; Kumar, P.; Gupta, C. K.; Indrayan, A. (M.L.N. Med. Coll., Allahabad, India). *Am. J. Proctol.* 26(2):75-86, 1975.

To assess the value of various clinical features in the diagnosis of abdominal tuberculosis, 141 cases of abdominal tuberculosis were analyzed clinically and histopathologically. Clinical features were assessed based on the hospital records of the cases admitted between 1967 and 1971. The histological features were determined using 70 specimens obtained from 57 cases. On the basis of the histological examinations, it was difficult to differentiate tuberculous from nonspecific lesions of the bowel. The presence of frank caseation, giant cells, epithelioid cells, and round cells are indicative of tuberculosis, but a microscopic picture similar to a nonspecific type of inflammation may also be present in tuberculous infections of the bowel. In the cases examined, a preoperative clinical diagnosis was given in only 39% of the patients; 8.6% were admitted with acute abdomen and 53.9% were diagnosed with intestinal obstruction. The incidence of the disease increased between 11 and 20 yr of age, peaking between 21 and 30 yr. The female:male ratio was 2.4:1. The commonest symptoms were generalized pain in and around the umbilical region, vomiting, and constipation; fever, weight loss, and anorexia were also common, and an associated pulmonary lesion was present in 5% of the patients. The most frequent physical signs were abdominal distension, tenderness, and abdominal lumps. Diarrhea was present in 17% of the cases, umbilical fistula in 2.5%, intraperitoneal abscess in 5%, and internal, perianal, and perirectal fistula in none. A diagnostic scoring system involving the allocation of points to parameters such as age, sex, symptoms, duration of symptoms, and signs has been devised; a score of 70 or above (maximum 100) is probably indicative of abdominal tuberculosis.

4653 MEGARECTUM IN CHILDREN: AN ANATOMICAL AND CLINICAL STUDY. *(Fr.)* Hata, Y.; Duhamel, B.; Pages, R.; Poirat, J. B.; Ripault, J. (Saint-Denis Hosp. Cent., France). *Ann. Chir. Infant.* 15(2):65-76, 1974.

Idiopathic megarectum was diagnosed and treated with anorectal sphincteromyectomy in 63 children. None of these patients had megarectum associated with anal imperforation, sacral agenesis, or spina bifida. Biopsies showed either abnormalities in the autonomic innervation of the juxta-anal segment of the rectum (19 cases), structural defects of the internal sphincter (15), or a combination of both abnormalities (19). No morphological abnormalities were detected in 10 cases. A statistical analysis of the date of onset of symptoms suggests that the disease is embryonic in origin. Sphincteromyectomy resulted in a complete cure in 54% of these children, a great improvement in 25%, some improvement in 10%, and no change in 11%. Most of the failures occurred in patients with abnormal innervation, and were later successfully treated with more extensive rectal surgery. The postoperative results were not dependent on age or on clinical or radiological features. This benign and effective method of diagnosis and treatment should be the initial surgical procedure in all patients with megarectum.

4654 AMMONIA AND UREA TRANSPORT BY THE EXCLUDED HUMAN COLON. *(E.)* Bown, R. L.; Gibson, J. A.; Fenton, J. C. B.; Snedden, W.; Clark, M. L.; Sladen, G. E. (St. Batholomew's Hosp., London, England). *Clin. Sci. Mol. Med.* 48(4):279-287, 1975.

Ammonia and urea transport across the colonic mucosa was studied in four subjects who had successfully undergone colonic exclusion for chronic hepatic encephalopathy. The colon was initially cleansed of fecal material and 24 hr later was perfused antiperistaltically with three isosmotic solutions containing 2 g/liter of the nonabsorbed marker polyethylene glycol. The peripheral venous blood was analyzed before perfusion and after the establishment of equilibrium conditions. In some cases, ammonium chloride or urea was added to the perfusion solutions or was infused i.v. Reduction of the luminal pH inhibited the net and unidirectional transport of ammonia from the lumen into the plasma, but the net absorption from high luminal concentrations persisted at low pH. Neither the addition of urea to the perfusate nor i.v. urea infusion produced a consistent increase in the colonic excretion of ammonia in the presence of amonnia-free

perfusates. In one subject, i.v. infusion of [^{15}N] ammonium chloride produced rapid labeling of the colonic effluent ammonia, and within 1 hr the specific enrichments of the ammonia in the effluent and in the arterial plasma were approximately equal. During perfusion with nitrogen-free solutions, only small amounts of urea appeared in the effluent, suggesting limited permeability of the colonic mucosa to urea. The results indicate that luminal acidification impairs net ammonia absorption from the colonic lumen and that ammonia either enters the lumen from the plasma or is generated within the lumen from urea or other sources. They also suggest that both ammonia and NH_4^+ diffuse across the colonic mucosa down electrochemical gradients and that the mucosa is considerably more permeable to ammonia than to NH_4^+. Exclusion of the colon appears to abolish the juxtamucosal conversion of urea into ammonia.

4655 RECTAL BIOPSY IN CLINDAMYCIN-ASSOCIATED COLITIS. AN ANALYSIS OF 23 CASES. *(E.)*
Sumner, H. W.; Tedesco, F. J. (Washington Univ. Sch. Med., St. Louis, Mo.). *Arch. Pathol.* 99(5):237-241, 1975.

The findings on rectal biopsy and proctoscope examinations in 23 cases of clindamycin-associated pseudomembranous colitis are summarized. The average age of the patients, 16 of whom were female, was 52 yr. The initial manifestations of colitis were diarrhea and abdominal pain, the average interval between the initiation of clindamycin therapy and onset of symptoms being 8 days. In 14 patients, clindamycin was administered orally (150 mg four times daily), in four it was administered i.v. (300-600 mg four times daily), and in five it was administered by both routes. The interval between the onset of symptoms and complete clinical resolution of the disease averaged 13 days; one patient died after 27 days. Therapy consisted of diphenoxylate hydrochloride with atropine sulfate and general supportive care. Stool cultures showed no growth of staphylococci or other enteric pathogens. On proctoscopic examination, discrete 2-5 mm raised plaques were seen adherent to an edematous, friable mucosa. Rectal biopsy showed pseudomembrane formation and inflammation of the underlying rectal mucosa. Necrosis of the surface epithelium was a frequent finding but no true ulcers were observed. The pseudomembrane was frequently observed to be dislodged from the mucosal surface. Vasculitis or thrombosis was not a feature in any case. Follow-up biopsies in four patients failed to show pseudomembranes and follow-up proctoscopic examinations showed no plaque-like lesions. While the pathologic and proctoscopic features of clindamycin-associated colitis are distinctive in the majority of cases, there are instances in which the disorder could be confused with ulcerative colitis. Proper diagnosis can be aided by awareness of the entity, repeated examinations when necessary, and communication between clinician and pathologist.

4656 BILE ACID EXCRETION AFTER PULL-THROUGH OPERATION FOR HIRSCHSPRUNG'S DISEASE. *(E.)*
Gaze, H.; Murphy, G. M.; Nelson, R.; Corkery, J. J.; Anderson, C. M. (Inst. Child Health, Univ. Birmingham, England). *Arch. Dis. Child.* 50(3):243-245, 1975.

To determine whether altered bile acid metabolism is involved in the production of chronic diarrhea after pull-through surgery for Hirschsprung's disease, the fecal and duodenal bile acids were studied in four patients suffering from diarrhea after such surgery. 3α-dehydrogenase and fluorimetry were used to determine the bile acid concentrations in three 24-hr fecal samples from each patient and in duodenal samples obtained after fasting and after i.v. pancreozymin administration. Fecal fat excretion was also determined, as was the bacterial content of the duodenal and gastric fluids. The fecal bile acids were elevated in all four children who had undergone the Soave pull-through operation in comparison with one child who had undergone colostomy for Hirschsprung's disease and 10 control children. In all five patients, the duodenal bile acid concentrations after pancreozymin stimulation were within normal limits. Bile acid analysis of the 'bile-rich' duodenal fluid obtained after pancreozymin stimulation in three of the pull-through patients indicated a marked reduction in the proportion of deoxycholic acid conjugates. In addition, two of these patients, who also had the highest fecal bile acid levels, showed an elevated ratio of glycine conjugated bile acids to taurine conjugated bile acids in the duodenal aspirate. The fecal fat levels and bacterial flora of the upper small intestine were normal in all cases. The results suggest colonic malabsorption of secondary bile acids in pull-through patients, but indicate that this is probably not the cause of the diarrhea and anal excoriation in these patients.

4657 A DOUBLE-BLIND COMPARISON OF INTENSIVE COURSE 5-FLUOROURACIL BY ORAL VS. INTRAVENOUS ROUTE IN THE TREATMENT OF COLORECTAL CARCINOMA. *(E.)*
Hahn, R. G.; Moertel, C. G.; Schutt, A. J.; Bruckner, H. W. (Mayo Clin. Mayo Found., Rochester, Minn.). *Cancer* 35(4):1031-1035, 1975.

The efficacy of p.o. and i.v. 5-fluorouracil (5-FU) administration in the treatment of colorectal carcinoma was studied using a randomized double-blind procedure. 5-FU was given p.o. (20 mg/kg/day) for 5 days to 47 patients with biopsy-proven metastatic adenocarcinoma of the colon and rectum; an i.v. dose of 13.5 mg/kg/day was given over the same period to 53 similar patients. To determine the effectiveness of the treatments, the patients were reevaluated at 5 and 10 weeks after the initiation of therapy. The blood levels of 5-FU were determined in 19 patients 15, 30, 45, 60, and 120 min posttreatment. There were no significant differences between the i.v.- and p.o.-treated patients in terms of toxic reactions to the drug; the gastrointestinal toxicity and stomatitis seen in each group did not require modification of the dosage and, although severe leukemia occurred in 31% of the patients, there were no drug-related deaths. With regard to the therapeutic results in the two groups, i.v. administration was associated with a nonsignificant increase in overall regression rate and a significant increase in the mean duration of response. Similar results were ob-

tained when both groups of patients were analyzed for specific response of the metastatic hepatic disease. I.v. administration was also associated with a more rapid decline in the 5-FU concentrations in the blood. In addition, while the blood concentration curves were comparable among the different patients receiving i.v., 5-FU, there was a striking variability among different patients receiving the drug p.o. The results suggest that, when indicated, 5-FU therapy is best provided by the i.v. route of administration.

4658 FIBER DEFICIENCY AND COLONIC DISORDERS. *(E.)* Reilly, R. W.; Kirsner, J. B. (Pritzker Sch. Med., Univ. Chicago, Ill.). *Am. J. Clin. Nutr.* 28(3):293-294, 1975.

4659 DIAGNOSIS OF NONSPECIFIC ULCERS OF THE COLON. *(Ger.)* Nuesch, H. J.; Blum, A. L.; Deyhle, P. (Triemli Munic. Hosp., Zurich, Switzerland). *Z. Gastroenterol.* 12(8):625-626, 1974.

4660 HISTOLOGIC CHANGES IN EXCLUDED AND NON-EXCLUDED INTESTINAL LOOPS INSERTED INTO THE URINARY TRACT. *(Cz.)* Kucera, J.; Malinsky, J.; Bana, J. (Med. Fac., Palacky Univ., Olomouc, Czechoslovakia). *Bratisl. Lek. Listy* 61(4):452-461, 1974.

4661 BLEEDING FROM THE LARGE INTESTINE. *(Ger.)* Pichlmayr, R.; Ziegler, H. (Med. Coll., Hannover, Germany). *Langenbecks Arch. Chir.* 337:577-584, 1974.

4662 X-RAY FINDINGS IN PEDIATRIC SURGICAL EMERGENCIES. (6) AGANGLIONOSIS OF THE WHOLE COLON. *(Jpn.)* Matsuyama, S.; Nagashima, K.; Suzuki, N.; Honda, R.; Isshiki, S.; Iwadare, J. (Gunma Univ. Sch. Med., Maebashi, Japan). *Kitakanto Med. J.* 24 (2):129-132, 1974.

4663 RECTAL POTENTIAL DIFFERENCE IN THE DIAGNOSIS OF ALDOSTERONE EXCESS. *(E.)* Beevers, D. G.; Morton, J. J.; Tree, M.; Young, J. (West Infirm., Glasgow, Scotland). *Gut* 16(1):36-41, 1975.

4664 STENOSIS OF THE COLON SECONDARY TO PANCREATITIS: REPORT OF A CASE. *(E.)* Theodoropoulos, G.; Archimandritis, A.; Kalos, A. (Med. Sch., Athens Univ., Greece). *Dis. Colon Rectum* 18 (2):158-160, 1975.

4665 A CASE OF BEHCET'S DISEASE WITH MULTIPLE INTESTINAL ULCERATIONS. *(Ger.)* Lehmann, H.; Stutte, H. J.; Zierott, G.; Schlaak, M. (Med. Clin. I, Univ. Kiel, Germany). *Dtsch. Med. Wochenschr.* 100(7):308-311, 1975.

4666 MAJOR COMPLICATIONS OF COLOSCOPY: BLEEDING AND PERFORATION. *(E.)* Geenen, J. E.; Schmitt, Jr., M. G.; Wu, W. C.; Hogan, W. J. (Racine Med. Clin., Milwaukee, Wis.). *Am. J. Dig. Dis.* 20 (3):231-235, 1975.

4667 INJURIES TO THE RECTUM IN DIAGNOSIS AND THERAPY. *(Ger.)* Decker, H. R.; Filler, D.; Feustel, H. (Surg. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Munch. Med. Wochenschr.* 117(8):291-292, 1975.

4668 EFFECT OF DRUGS ON MOTILITY OF THE ILEOCECAL SPHINCTER IN ACUTE OBSTRUCTIONS OF THE LARGE INTESTINE. *(Ger.)* Willmen, H. R.; Kogel, H.; Schwilden, E. D. (Med. Fac., Rhenish-Westphalian Eng. Coll., Aachen, Germany). *Int. J. Clin. Pharmacol.* 9(4):314-320, 1974.

4669 INTESTINAL OBSTRUCTION AS A COMPLICATION OF LONG-TERM ANTICOAGULANT THERAPY. *(Fr.)* Sarris, M.; Georgoulis, J.; Gatos, M.; Zervos, S. N. (Hippokrateion Hosp., Athens, Greece). *Schweiz. Med. Wochenschr.* 102(42):1480-1482, 1974.

4670 DIVERTICULOSIS AND DIVERTICULITIS OF THE COLON. *(Fr.)* Girard, M. (no affil.). *J. Med. Lyon* 55(1271):849-861, 1974.

4671 DIVERTICULOSIS OF THE LARGE INTESTINE. CLINICAL STUDY. *(Pol.)* Kaszewska-Jablonska, I.; Gorska-Dubowik, M.; Bielecki, M.; Czechlowski, B. (Acad. Med., Gdansk, Poland). *Pol. Tyg. Lek.* 27(48):2105-2107, 1974.

4672 PARENTERAL PRE- AND POSTOPERATIVE TREATMENT OF PATIENTS WITH INTESTINAL FISTULAS. *(Ger.)* Dudziak, R. (Clin., Univ. Frankfurt/Main, Germany). *Langenbecks Arch. Chir.* 337:141-145, 1974.

4673 FISTULAS IN THE SMALL INTESTINE, LARGE INTESTINE AND ANORECTUM. *(Ger.)* Stelzner, F. (Cent. Surg., Univ. Frankfurt/Main, Germany). *Langenbecks Arch. Chir.* 337:131-134, 1974.

4674 THE LOCATION OF ANAL GLANDS IN MAN IN RELATION TO THE CIRCUMFERENCE OF THE ANAL CANAL: THEIR IMPORTANCE IN THE PATHOGENESIS OF ANORECTAL FISTULAS. *(Ger.)* Krakovic, M. (Cent. Surg., Univ. Frankfurt/Main, Germany). *Langenbecks Arch. Chir.* 336(2):141-154, 1974.

4675 A STUDY OF EXPERIMENTAL COLOVESICAL FISTULA. *(E.)* Heiskell, C. A.; Ujiki, G. T.; Beal, J. M. (Northwest. Univ. McGraw Med. Cent., Chicago, Ill.). *Am. J. Surg.* 129(3):316-318, 1975.

4676 COLONOSCOPIC POLYPECTOMY. *(Fr.)* Chabanon, R. (Edouard Herriot Hosp., Lyon, France). *Rev. Fr. Gastroenterol.* (100):53-55, 58, 1974.

4677 ARTERIOGRAPHIC DIAGNOSIS OF A JUVENILE COLON POLYP. *(E.)* Lynch, W. A. (Hartford Hosp., Conn.). *Am. J. Gastroenterol.* 63(2):166-168, 1975.

4678 FAMILIAL POLYPOSIS OF COLON AND RECTUM. *(Ger.)* Hantschmann, N.; Nemsmann, B. (Surg. Clin., Univ. Kiel, Germany). *Dtsch. Med. Wochenschr.* 99(10):453-457, 1974.

4679 GARDNER'S SYNDROME AND FIBROMATOSIS: REVIEW OF THE PROBLEM AND REPORT OF A CASE. *(E.)* Shons, A. R.; Estrin, J.; Najarian, J. S. (Univ. Minnesota Hosp., Minneapolis). *Dis. Colon Rectum* 18(2):128-133, 1975.

4680 LYMPHOID POLYPOSIS OF THE TERMINAL ILEUM AND COLON: A SOURCE OF CLINICAL ERROR. *(E.)* LoGerfo, F. W.; Mueller, R. F. (Dep. Surg., Univ. Maryland, Baltimore). *Am. Surg.* 41(3):179-180, 1975.

4681 ENTEROCOLITIS IN HIRSCHSPRUNG'S DISEASE. *(Dut.)* Lippens, R. J. J.; Van Putten, W. J. (Emma-Pediatr. Hosp., Amsterdam, Netherlands). *Maandschr. Kindergeneeskd.* 41(7/8):239-246, 1973.

4682 GARDNER'S SYNDROME. *(Jpn.)* Tanaka, M.; Hayano, I.; Okubo, T.; Nishioka, K. (Med. Sch., Keio Univ., Japan). *Jpn. J. Clin. Radiol.* (19): 781-785, 1974.

4683 PEUTZ-JEGHER'S SYNDROME. *(Ser.)* Jovanovic, B.; Glidzic, V.; Stefanovic, Z. (Med. Fac., Univ. Belgrade, Yugoslavia). *Srp. Arh.* 102(6):479-484, 1974.

4684 CECAL ULCER CAUSED BY CYTOMEGALOVIRUS FOLLOWING KIDNEY TRANSPLANTATION. *(Ger.)* Clavadetscher, P.; Sulser, H.; Linder, E.; Tschen, H.; Deyhle, P. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(8):625, 1974.

4685 MUCOPOLYSACCHARIDES IN THE EPITHELIUM OF NORMAL MUCOSA IN THE LARGE INTESTINE AND IN RECTAL AND RECTOSIGMOID CANCER. *(Rus.)* Lazarev, I. M. (Moldavian Res. Inst. Oncol., Kishenev, USSR). *Lab. Delo* (6):335-338, 1974.

4686 PRIMARY CARCINOMA OF THE VERMIFORM APPENDIX. *(E.)* Pendse, A. K.; Dandia, S. D.; Narula, I. M. S.; Ramdeo, I. N. (Sardar Patel Med. Coll., Bikaner, India). *Indian J. Surg.* 36(7):285-286, 1974.

4687 ANTI-TUMOUR IMMUNOREACTIVITY IN COLONIC CARCINOMA. A GUIDE TO EARLY RECOGNITION OF RECURRENCE. *(E.)* Nairn, R. C.; Rollo, A. J.; Nind, A. P. P.; Rolland, J. M.; Guli, E. P.; Pihl, E. A. V.; Stevens, D. P. (Dep. Pathol. Immunol., Monash Univ., Melbourne, Australia). *Aust. NZ J. Med.* 4(6):531-534, 1974.

4688 PREDISPOSING FACTORS IN CARCINOMA OF THE COLON. *(E.)* Sherlock, P.; Lipkin, M.; Winawer, S. J. (Mem. Sloan-Kettering Cancer Cent., New York, N.Y.). *Adv. Intern. Med.* 20:121-150, 1975.

4689 SUTURE-LINE RECURRENCE IN CARCINOMA OF THE COLON AND RECTUM. *(E.)* Labow, S. B.; Salvati, E. P.; Rubin, R. J. (Muhlenberg Hosp., Plainfield, N.J.). *Dis. Colon Rectum* 18(2):123-125, 1975.

4690 IMMUNITY IN COLONIC TUMOR PATIENTS AFTER OPERATION: DETERMINATION BY LEUKOCYTE-MIGRATION INHIBITION. *(E.)* House, A. K.; Wisniewski, S.; Woodlings, T. L. (Perth Med. Cent., Univ. West. Australia, Shenton Park). *Dis. Colon Rectum* 18(2):100-106, 1975.

4691 COMPLETE INVAGINATION OF THE VERMIFORM APPENDIX WITH VILLOUS ADENOMA, INTUSSUSCEPTING TO THE SPLENIC FLEXURE OF THE COLON. *(E.)* Ho, L.; Rosenman, L. D. (Mt. Zion Hosp. Med. Cent., San Francisco, Calif.). *Surgery* 77(4):505-506, 1975.

4692 MALIGNANT TUMORS OF THE ANAL CANAL. *(Fr.)* Stalport, J.; Boddaert, J.; Rase, M.; Quertillier, D.; Toussaint, M. (Reine Astrid Clin., Huy, Belgium). *Acta Gastroenterol. Belg.* 37(6):291-301, 1974.

4693 OBSTRUCTION AND PERFORATION AS COMPLICATIONS OF LARGE BOWEL CANCERS: ISCHEMIC MODIFICATIONS OF THE DISTENDED COLONIC WALL. *(Fr.)* Saegesser, F.; Tabrizian, N. (Univ. Surg. Serv., Lausanne, Switzerland). *J. Med. Strasbourg* 5(4):299-311, 1974.

4694 ADENOCARCINOMA OF THE COLON PRESENTING AS Clostridium septicum CELLULITIS OF THE LEFT THIGH. *(E.)* Poretz, D. M.; Wood, L.; Park, C. (Fairfax Hosp., Falls Church, Va.). *South Med. J.* 67(7):862-864, 1974.

4695 ISCHEMIC LESIONS OF THE DISTENDED COLON. A COMPLICATION OF OBSTRUCTIVE COLORECTAL CANCER. *(E.)* Saegesser, F.; Sandblom, P. (Univ. Laussane Sch. Med., Switzerland). *Am. J. Surg.* 129 (3):309-315, 1975.

4696 ANORECTAL MELANOMA. *(E.)* Balthazar, E. J.; Javors, B. (New York Med. Coll., N.Y.). *Am. J. Gastroenterol.* 63(1):79-83, 1975.

4967 UROLOGIC COMPLICATIONS AFTER OPERATIONS FOR ANORECTAL CANCER, WITH AN EVALUATION OF PREOPERATIVE INTRAVENOUS PYELOGRAPHY. *(E.)* Kramhoft, J.; Kronborg, O.; Backer, O. G.; Sprechler, M. (Bispeberg Hosp., Copenhagen, Denmark). *Dis. Colon Rectum* 18(2):118-122, 1975.

4698 PRE-RENAL FAILURE DUE TO VILLOUS ADENOMA OF THE COLON. *(Ger.)* Lange, H. H.; Renner, E. (Cologne-Merheim Munic. Hosp., Germany). *Dtsch. Med. Wochenschr.* 100(6):246-250, 1975.

4699 CARCINOMA OF THE COLON AND RECTUM: ADVANCES IN ITS EPIDEMIOLOGY, ETIOLOGY, AND DIAGNOSIS. *(Ger.)* Feurle, G. (Med. Polyclin., Univ. Heidelberg, Germany). *Med. Welt.* 69(43):1734-1744, 1974.

4700 THE IRRITABLE COLON. A RETROSPECTIVE STUDY OF 217 CASES. *(Ger.)* Ruiz De Aguiar Gonzalez, A.; Audibert Mena, L. (San Carlos Hosp., Madrid, Spain). *Munch. Med. Wochenschr.* 116(38):1641-1646, 1974.

4701 CLINDAMYCIN-ASSOCIATED COLITIS. *(E.)* Unger, J. L.; Penka, W. E.; Lyford, C. (Univ. Kansas Med. Cent., Kansas City). *Am. J. Dig. Dis.* 20(3):214-222, 1975.

4702 CLINDAMYCIN COLITIS FOLLOWING CLINDAMYCIN THERAPY: REPORT OF A CASE. *(E.)* Butsch, J. L.; Al-Humadi, A. H.; Alford, J. E. (Buffalo Gen. Hosp., N.Y.). *Dis. Colon Rectum* 18(2):152-157, 1975.

4703 ISCHEMIC COLITIS WITH PERFORATION INTO THE GALLBLADDER. *(E.)* Wilk, P. J. (Mt. Sinai Hosp., City Univ. New York, N.Y.). *Mt. Sinai J. Med. NY* 42(2):119-126, 1975.

4704 GRANULOMATOUS COLITIS: THERAPY. *(Sp.)* Meeroff, M. (Gregorio Araoz Alfaro Polyclin., Buenos Aires, Argentina). *Galicia Clin.* 45 (10):935-939, 1973.

4705 ISOLATED TUBERCULOSIS OF APPENDIX. *(E.)* Mittal, V. K.; Khanna, S. K.; Gupta, N. M.; Aikat, M. (Postgrad Institute Med. Educ. Res., Chandigarh, India). *Am. Surg.* 41(3):172-174, 1975.

4706 EXAMPLE OF PNEUMATOSIS CYSTOIDES INTESTINALIS. *(Jpn.)* Otawa, K.; Yamasaki, M.; Tashiro, S. (no affil.). *Jpn. J. Clin. Radiol.* (19): 715-721, 1974.

4707 VILLOUS ADENOMA OF THE CECUM. *(E.)* Kilam, S. K.; Costopoulos, L. B.; Yakimets, W. W. (Mem. Univ. Newfoundland, St. John's). *Can. J. Surg.* 18(3):214-215, 218, 1975.

4708 HIRSCHSPRUNG'S DISEASE: ITS AETIOLOGY, PATHOGENESIS AND DIFFERENTIAL DIAGNOSIS. *(E.)* Meier-Ruge, W. (Exp. Pathol. Histochem. Sect., SANDOZ Ltd., Basel, Switzerland). *Curr. Top. Pathol.* 59:131-179, 1974.

See also, 4212, 4236, 4237, 4238, 4239, 4240, 4241, 4243, 4244, 4284, 4395, 4431, 4549, 4579, 4585, 4597, 4721, 4994, 5043, 5084, 5131, 5155, 5156, 5163, 5180, 5185, 5187, 5192.

LARGE INTESTINE

Ulcerative Colitis

4709 EFFECT OF A CHEMICALLY DEFINED LIQUID ELEMENTAL DIET ON COMPOSITION AND VOLUME OF ILEAL FISTULA DRAINAGE. *(E.)* Hill, G. L.; Mair, W. S. J.; Edwards, J. P.; Morgan, D. B.; Goligher, J. C. (Gen. Infirm., Leeds, England). *Gastorenterology* 68 (4):676-682, 1975.

The effect of an elemental diet on the quantity and composition of the ileal fistula drainage was studied in 10 patients with well-established ileostomies. During the first 4 days of the study period, the patients, all of whom were in good health, consumed a "normal" control diet. At the end of this period, the patients were put on a standard elemental diet which supplied the same amount of calories as the control diet. After 8 days on this diet, the patients were put on a 3-day regimen in which 2000 ml of water were taken in addition to the elemental diet. The water content in the feces and ileostomy fluid, the sodium and potassium contents in the urine and ileostomy fluid, and the urinary and ileostomy fluid volumes were measured throughout the study. The mean daily fecal output during the control period was 570 g; during the administration of the elemental diet the fecal output fell to 441 g ($P > 0.02$). In addition the output of sodium in the ileostomy fluid fell from 62.7 to 3.41 mEq ($P > 0.02$) and the output of potassium fell from 5.2 to 2.9 mEq ($P > 0.001$) during administration of the elemental diet, and the concentration of sodium fell from 104.7 to 80.2 mEq/liter ($P > 0.01$). The proportion of water in the ileostomy fluid rose from 90.9 to 94.2% ($P > 0.001$) during this period. When the patients consumed an additional 2 liters of water with the elemental diet, the only

significant change observed was a rise in the daily urinary output. The results indicate that under controlled conditions, maintenance on an elemental diet leads to decreases in the gastrointestinal output of fluid, sodium, and potassium, and increases in the water content. These changes were, however, less pronounced than previous reports would suggest.

4710 HERNIATION OF MUCOSAL EPITHELIUM INTO THE SUBMUCOSA IN CHRONIC ULCERATIVE COLITIS. *(E.)* Dyson, J. L. (City Hosp., Nottingham, England). *J. Clin. Pathol.* 28(3):189-194, 1975.

The mechanism of glandular herniation on chronic ulcerative colitis and its relation to carcinoma was studied in 27 patients. They had had colitis from 3-24 yr and 8 had one or more carcinomas. All slides from biopsy specimens in each case were examined. Of 27 cases, 11 showed crypts, cysts, or abscess walls of glandular epithelium in the submucosa. This misplaced epithelium occurred in all parts of the colon, either in islands or as downgrowths continuous with the mucosal glands. When surgery was performed during quiescent periods of ulceration, the mucosal downgrowth was seen usually to occur where the muscularis mucosae had been breached and replaced by a lymphoid aggregate. Slight glandular projection into lymphoid aggregates occurred in almost all cases, but projection into the submucosa was relatively rare. Histological widening of the muscularis mucosae occurred in 8 of 11 patients with mucosal herniation and in 6 with no herniation. There was glandular crowding in 9 cases, 5 of which were associated with herniation. The degree of present inflammation or part ulceration did not bear any relation to herniation. None of the patients with carcinoma had ulcerative colitis for less than 8 yr. Two patients had the disease for 14 and 15 yr, but did not develop carcinoma. Herniation and carcinoma developed independently of each other. It is concluded that herniation is probably due to sustained contraction of the muscularis mucosae, and is not in itself an indication os potential malignancy.

4711 IMPORTANCE OF EARLY INTENSIVE TREATMENT IN SEVERE ATTACKS OF ULCERATIVE COLITIS. *(E.)* Barany, F.; Lindholmer, B. (St. Erik's Hosp., Stockholm, Sweden). *Rend. Gastroenterol.* 6(3): 174-178, 1974.

The clinical course in 41 cases of emergency surgery associated with ulcerative colitis is reviewed to determine whether emergency operations in this disease are avoidable. The first symptoms noted by the patient were slight in 12 cases, moderate in 22, and severe in seven. The mean amount of time elapsed between the onset of symptoms and the first visit to a physician was 18.1 days, while the mean duration between first visit to a doctor and diagnosis and emergency surgery averaged 35.1 days. Among the 41 emergency cases, the diagnosis of ulcerative colitis had been made at an earlier attack in 17 cases, while in 24 cases, surgery was required at the first attack. The data suggest that emergency operations in ulcerative colitis are avoidable if: attacks of this disease are treated immediately upon diagnosis; moderate to severe attacks are treated with high doses of corticosteroids, not sulfasalazine and topical steroids; surgery is performed without delay when a few days of intensive corticoid therapy was not followed by definite improvement of the general condition and bowel inflammation; and anticholinergic and ganglion-blocking agents are avoided during active colitis.

4712 INFLAMMATORY DISEASE OF THE COLON: ULCERATIVE COLITIS AND CROHN'S COLITIS. *(E.)* Ament, M. E. (Univ. California Los Angeles Cent. Health Sci.). *J. Pediatr.* 86(3):322-334, 1975.

4713 NONSPECIFIC ULCERATIVE COLITIS. *(E.)* Pimparkar, B. D. (K.E.M. Hosp., Bombay, India). *Indian Pract.* 27(12):593-603, 1974.

4714 PROTEINS AND GLYCOPROTEINS IN GASTRIC JUICE FROM PATIENTS WITH ULCERATIVE COLITIS. *(Pol.)* Chacaj, W.; Kuczynska-Sekieta, K.; Ceislinska, K.; Malolepszy, J.; Suchnicka, R.; Dec, J. (Acad. Med., Wroclaw, Poland). *Pol. Tyg. Lek.* 29(37): 1579-1531, 1974.

4715 STUDY OF THE LEUKOCYTE AGGLUTINATION REACTION IN PATIENTS WITH NONSPECIFIC ULCERATIVE COLITIS UNDER INPATIENT AND OUTPATIENT CONDITIONS. *(Rus.)* Shakhbazian, A. A. (Erevan Med. Inst., USSR). *Zh. Eksp. Klin. Med.* 14(3):45-50, 1974.

4716 CHANGES IN THE INTESTINAL FLORA IN NONSPECIFIC ULCERATIVE COLITIS. *(Rus.)* Shakhbazian, A. A. (Yerevan Med. Inst., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (12):108-109, 1975.

4717 EMERGENCY AND URGENT OPERATIONS FOR ULCERATIVE COLITIS. THE PROCEDURE OF CHOICE. *(E.)* Binder, S. C.; Miller, H. H.; Deterling, Jr., R. A. (Tufts Univ. Sch. Med., Boston, Mass.). *Arch. Surg.* 110(3):284-289, 1975.

4718 RECTAL CONSERVATION IN THE SURGICAL TREATMENT OF ULCERATIVE COLITIS. *(E.)* Aylett, S. G. (Westminster Hosp., London, England). *Arch. Fr. Mal. App. Dig.* 63(7):585-587, 1974.

4719 COLECTOMY AND ANASTOMOSIS FOR INFLAMMATORY BOWEL DISEASE. *(E.)* Ritchie, J. K. (St. Mark's Hosp., London, England). *Arch. Fr. Mal. App. Dig.* 63(7):588, 1974.

4720 ILEO-RECTAL ANASTOMOSIS FOR INFLAMMATORY BOWEL DISEASE IN CHILDREN. *(E.)* Nixon, H. H. (Hosp. Sick Child., London, England). *Arch. Fr. Mal. App. Dig.* 63(7):590-591, 1974.

4721 COMPARISON OF BOWEL FUNCTION AFTER ILEO-RECTAL ANASTOMOSIS FOR ULCERATIVE COLITIS AND COLONIC POLYPOSIS. *(E.)* Newton, C. R.; Baker, W. N. W. (St. Mark's Hosp., London, England). *Arch. Fr. Mal. App. Dig.* 63(7):581-593, 1974.

See also, 4647, 4665, 4953, 5057, 5071, 5160, 5161, 5162, 5165, 5181, 5184.

PANCREAS

4722 FURTHER EVALUATION OF TOTAL PANCREATECTOMY. *(E.)* Pliam, M. B.; ReMine, W. H. (Mayo Clin., Rochester, Minn.). *Arch. Surg.* 110(5):506-512, 1975.

The results of 28 total pancreatectomies performed from 1969-1973 were compared with those of 37 total pancreatectomies performed from 1942-1969. The entire series of 64 patients included 39 men and 25 women, aged 28-77 yr. Among the 40 patients who underwent total pancreatecomy for cancer, 24 had pancreatic carcinoma, 2 islet cell carcinoma, 9 ampullary carcinoma, 3 cystoadenocarcinoma, 1 duodenal carcinoma, and 1 rhabdomyosarcoma. At 6 months, 1 yr, and 3 yr, the survival for the early series was 78, 50, and 17% resp. For the recent series, corresponding rates were 80, 56, and 44%. While not statistically significant, these differences suggest survival rates may be improving over the early series. The difference in survival of cancer and noncancer patients was significant at 1 yr (53 *versus* 83%) and was more striking at 3 yr (25 *versus* 83%). The longest survival was 29 yr 3 months, after total pancreatecomty for islet cell tumor. The longest survival in patients with adenocarcinoma of the pancreas was 6 yr 8 months; in patients with ampullary carcinoma, 6 yr 6 months; in patients with cystadenocarcinoma, 4 yr 9 months; and patients with duodenal carcinoma, 10 months. Of the 5 patients who underwent total pancreatectomy for chronic pancreatitis, the longest survival was 8 yr. In the recent series, wound infection decreased from 33 to 11%, gastrointestinal tract bleeding from 28 to 15%, subphrenic abscess from 20 to 11%, and dumping syndrome from 14 to 7%. The incidence of diabetic ketoacidosis was only 3% for the entire group and did not differ between the early and recent series. However, the frequency of difficult diabetes in the early postoperative period decreased from 25% in the early series to 15% in the recent series. Recognition of the fact that relatively small doses of insulin are necessary to control diabetes in these patients probably accounts for the improvement in management. Major gastrointestinal tract bleeding decreased from 20% to 0 through the use of more extensive gastric resection. These recent results are more favorable than those reported for a group of comparable patients undergoing standard Whipple procedures for malignant lesion at the same institution.

4723 RELATIONSHIP BETWEEN ALCOHOLISM AND PANCREATIC INSUFFIENCY. *(E.)* DiMagno, E. P.; Malagelada, J. R.; Go, V. L. M. (Mayo Clin., Rochester, Minn.). *Ann. NY Acad. Sci.* 252:200-207, 1975.

Basal pancreastic trypsin and lipase outputs in response to a maximal stimulatory dose of cholecystokinin-pancreozymin (CCK-PZ, 0.25 CHR U/kg/min i.v.) were measured in 17 patients (11 men and 6 women, aged 35-71 yr) with chronic alcoholic pancreatitis and in 21 healthy men (aged 21-56 yr). In the controls, maximal CCK-PZ stimulation produced a 2.84-fold increase in lipase output and a 3.5-fold increase in trypsin output over basal levels. No significant increase above basal enzyme levels occurred in chronic pancreatitis. The ratio of lipase to trypsin output in both the basal and stimulatory state was significantly higher in controls than in chronic pancreatitis patients. All 5 patients with a greater than 20-yr history of alcoholism had lipase and trypsin outputs less than 10% of normal maximal values; of the 12 patients with alcoholism of 10-20 yr duration, 11 had below-normal lipase outputs. All patients with greater than 20 yr of alcoholism had pancreatic calcification, steatorrhea, and diabetes. Although 8 patients with a shorter duration of alcoholism had steatorrhea and 7 had diabetes or steatorrhea, only 4 had all of these. The data suggest that lipase secretion is a more reliable predictor of exocrine secretory reserve function than is trypsin secretion and that steatorrhea is a late but inevitable sequel of a progressive decline of pancreatic exocrine enzyme outputs.

4724 SELECTIVE ARTERIOGRAPHY OF THE CELIAC-MESENTERIC ARTERY IN THE DIAGNOSIS AND ASSESSMENT OF OPERABILITY OF PANCREATIC NEOPLASMS. *(It.)* Tateo, R.; Romani, F.; Meinero, M.; Zagnoli, P.; Galasso, U. (Inst. Spec. Pathol. Clin. Propedeut., Univ. Modena, Italy). *Chir. Gastroenterol.* 8(3):300-317, 1974.

4725 ADRENOCORTICAL TISSUE LOCATED IN THE PANCREAS: REPORT OF A CASE. *(It.)* Fortis, P. A.; Longhino, C.; Postiglione, G. (Maggiore, Hosp., Bergamo, Italy). *Osp. Ital. Chir.* 27(1):25-34, 1974.

4726 SURGICAL TREATMENT OF POSTOPERATIVE COMPLICATIONS AND LONG-TERM RESULTS IN PANCREATIC PSEUDOCYSTS. *(Cs.)* Liskova. M. (Dep. Abdom. Thorac. Surg., UVN, Prague, Czechoslovakia). *Rozhl. Chir.* 53(7):454-459, 1974.

4727 ECHOTOMOGRAPHY IN THE DIAGNOSIS OF PANCREATIC PSEUDOCYSTS. *(Fr.)* Levrat, M.; Descos, L.; Pillon, M.; Paliard, P.; Roussille, M.; Duquesnel, C. (Eduoard Herriot Hosp., Lyon, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(5):367-375, 1974.

4728 PSEUDOCYSTS OF THE PANCREAS. REVIEW OF 71 CASES. *(E.)* van Heerden, J. A.; Re-Mine, W. H. (Mayo Clin., Rochester, Minn.). *Arch. Surg.* 110(5):500-505, 1975.

4729 PANCREATIC CYST COMPLICATED BY MAJOR ARTERIAL EROSION AND GASTROINTESTINAL HAEMORRHAGE. *(E.)* Wilson, W. F.; Wilkie, R. C.; Ewing, M. R. (R. Melbourne Hosp., Australia). *Aust. NZ J. Surg.* 45(1):85-90, 1975.

4730 ALCOHOL AND THE PANCREAS. *(E.)* Sarles, H. (Dig. Pathol. Res. Unit, INSERM, Marseille, France). *Ann. NY Acad. Sci.* 252:171-182, 1975.

4731 PANCREATOJEJUNOSTOMY IN PANCREATODUODENECTOMY. *(Rus.)* Linchenko, I. F. (Stavropol Med. Inst., USSR). *Vestn. Khir.* 114(3):33-36, 1975.

4732 PANCREATIC TRAUMA. *(E.)* Mosavy, S. H.; Vakshuri, P.; Tadaion, A. (Isfahan Univ. Sch. Med., Iran). *Abdom. Surg.* 17(5):113, 115-116, 1975.

4733 ANATOMOCLINICAL OBSERVATIONS ON TWO CASES OF ABERRANT PANCREAS. *(Rum.)* Popvici, G. I.; Mocanu, A.; Onofrei, T.; Popovici, G. G. (Dep. Surg., United Hosp., Bacau, Rumania). *Rev. Med. Chir.* 78(1):165-168, 1974.

4734 THE STUDY OF PANCREATIC FUNCTION ON ONE CASE OF CYSTIC FIBROSIS. *(Sp.)* Figueroa, C.; Larrain, F.; Danus, O.; Urbina, A. M. (Roberto del Rio Hosp., Santiago, Chile). *Rev. Pediat. Santiago* 17(1/2):7-11, 1974.

4735 EXCRETION OF ZINC IN PANCREATIC JUICE IN THE SECRETIN-PANCREOZYMIN TEST. *(Ger.)* Persigehl, M.; Kasperek, K.; Loffler, A.; Feinendegen, L. E. (Med. Polyclin., Univ. Bonn, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:539-541, 1974.

4736 DISSOCIATION OF PANCREATIC ENZYME SECRETION AND GALLBLADDER EMPTYING AFTER INTRADUODENAL PERFUSION OF CALCIUM. *(Ger.)* Holtermuller, K. H.; Malagelada, J. R.; Go, V. W. L. (Med. Clin., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:537-539, 1974.

See also, 4188, 4199, 4213, 4214, 4233, 4245, 4247, 4248, 4249, 4251, 4252, 4253, 4254, 4256, 4257, 4768, 4920, 4948, 5068, 5094.

PANCREAS

Pancreatitis

4737 CHRONIC CALCIFYING PANCREATITIS IN BRAZIL: MORPHOLOGICAL STUDIES. *(E.)* Nogueria, C. E. D.; Dani, R.; Brandao, H. S.; Nunes, A. (Fac. Med., Univ. Minas Gerais, Belo Horizonte, Brazil). *Rend. Gastroenterol.* 6(3):157-164, 1974.

A morphological study of 38 cases of chronic calcifying pancreatitis showed that the calcifications were diffuse in 33 patients, localized in the head in 4, and localized in the body and tail in 1. Stenosis of the cephalic segment of the duct was observed in all 21 patients submitted to an operative pancreatography; 4 of these patients had a second stenosis in the body and in the tail. Retention cysts were observed in the pancreas of 9 subjects and pseudocysts in 6. Diffuse and heavy induration was grossly present at operation in the 33 patients with diffuse calcifications. In 3 cases with segmental calcifications the fibrosis was more pronounced in the calcified area but was evident in the whole pancreas; in 2 cases with stones in the head of the pancreas, only light sclerosis was seen in the body and the tail. Two patients showed intense edema of the whole pancreas and 15 presented with severe sclerosis of the peripancreatic tissue. The diameter of the common duct, studied in 33 cases by i.v. cholangiography and/or operative radiomanometry, was 0.5-2.3 cm (mean 1.06 $\pm$ 0.08). Diffuse portal hypertension was detected in $\frac{3}{3}$ patients and segmentary portal hypertension due to thrombosis of the splenic vein was observed in 1 case. Enlargement of the head of the pancreas was responsible for duodenal obstruction in 3 cases. Microscopic examination of 5 autopsy cases and 19 surgical specimens showed a high incidence of parenchymal atrophy (65%), regression to the canalicular status (70%), islet atrophy (80%), dilation of the ducts (80%), protein plugs (50%), and fibrosis (83%). In 2 patients carcinoma of the pancreas was found 3 and 5 yr after diagnosis of CCP.

4738 PANCREATICO-JEJUNOSTOMY IN CHRONIC PANCREATITIS. AN APPRAISAL OF 29 CASES. *(E.)* Arnesjo, B.; Ihse, I.; Kugelberg, C.; Tylen, U. (Dep. Surg., Univ. Lund, Sweden). *Acta Chir. Scand.* 141: 139-148, 1975.

Pancreatico-jejunostomy (end-to-side after resection of the pancreatic tail or side-to-side following longitudinal cleaving of the pancreatic duct) was performed on 29 patients (22 men and 7 women, aged 14-61 yr) with chronic relapsing or acute relapsing pancreatitis. A follow-up study ranging from 6 months to 10 yr is presented. The patients were allotted

to 3 groups according to the severity and extent of the inflammatory involvement at operation. The diagnostic findings obtained at the preoperative evaluation correlated well with these groups. In some patients, however, the inflammatory process was mainly confined to the ductal system, as revealed by pancreatography, while in others the changes were most marked in the parenchyma, as found at histologic examination. Twenty-one caudal pancreatico-jejunostomies and 8 longitudinal anastomoses were performed. Overall results were excellent in 20 patients, good in 6, fair in 3, and poor in none. The rate of postoperative complications was low: no mortality, 1 spontaneously healed anastomosis insufficiency, 2 cases of subphrenic abscess, and 5 cases of postoperative diabetes. Only one patient had a recurrent attack of acute pancreatitis postoperatively. Reanastomosis was successfully performed. It is concluded that pancreatico-jejunosotomy, based on findings at pancreatography, may reduce the risk of serious, late complications of the disease.

4739 EVALUATION OF PROPHYLACTIC ANTIBIOTICS IN ACUTE PANCREATITIS. *(E.)* Howes, R.; Zuidema, G. D.; Cameron, J. L. (John Hopkins Med. Inst., Baltimore, Maryland). *J. Surg. Res.* 18(2):197-200, 1975.

A prospective randomized study was performed to compare the incidence of septic complications in patients with acute pancreatitis treated with antibiotics and in patients receiving no antibiotic therapy. Of 48 patients randomized to the antibiotic group, 44 received ampicillin (1 g every 6 hr for 5 days) and 4 received lincomycin (600 mg i.v. every 8 hr followed by 500 mg p.o. every 6 hr for 5 days). Forty-seven patients received no antibiotic therapy. Age, race, sex distributions were similar within the 2 groups, and in each group alcohol consumption was the most common etiology of the pancreatitis. Average initial serum amylase values were 392 Caraway U/100 ml in the antibiotic group and 365 Caraway U in the no antibiotic group. The amylase level remained elevated for an average of 2 days in both groups. Five septic complications developed in the antibiotic group, and 6 in the untreated group. In the antibiotic group 3 patients developed pneumonia and 2 developed pancreatic abscess. An organism was grown in only 2 of these 5 cases and was an ampicillin-resistant Klebsiella. In the no antibiotic group there were 4 pneumococcal pneumonias, all sensitive to ampicillin, and 1 pancreatic abscess secondary to ampicillin-resistant Klebsiella and Enterobacter. One patient given no antibiotics developed a septicemia, but its source was not found. One patient in the antibiotic group developed an ampicillin allergy and was switched to lincomycin. The overall incidence of septic complications in the two groups did not differ significantly. These results show no advantage of using antibiotics in the treatment of acute pancreatitis until there is clinical or bacteriological evidence of sepsis.

4740 CONSISTENT PROTECTION FROM PANCREATITIS IN CANINE PANCREAS ALLOGRAFTS TREATED WITH 5-FLUOROURACIL. *(E.)* Castellanos, J.; Manifacio, G; Toledo-Pereyra, L. H.; Shatney, C. H.; Lillehei, R. C. (Dep. Surg., Univ. Minnesota, Minneapolis). *J. Surg. Res.* 18(36):305-311, 1975.

The ability of 5-fluorouracil (5-FU) to protect against pancreatitis following pancreas transplantation was studied in 5 groups of adult mongrel dogs. A no-transplant control group (10 dogs) was treated with azathioprine (5.0 mg/kg/day i.v. for 3 days, then 2.5 mg/kg/day p.o) plus 5-FU (15 mg/kg/day i.v. on day 1, then 7.5 mg/kg/day p.o. for 1 week). Control transplanted dogs (6) received no immunosuppressive treatment. Seven transplanted dogs were given azathioprine only; 8 of 14 recipient dogs treated with both azathioprine and 5-FU (15 mg/kg/day the day of the transplant and then 7.5 mg/kg/day for 1 week) received a pancreas from donors treated with 5-FU (15 mg/kg/day) for 2 days and 1 hr before surgery, while the remaining dogs received a pancreas from a donor treated with 5-FU only 1 hr before surgery. Dogs that received no immunsuppression lived an average of 5.5 days. When azathioprine was added to the treatment, survival increased to an average 11.1 days. Only when 5-FU was added to the minimal immunosuppression treatment did the transplanted dogs live for periods up to 4 weeks. Results were best in dogs receiving a pancreas from donors given 5-FU for 2 days before transplantation. The 4 recipient groups did not differ with respect to postoperative total protein, albumin, or blood glucose levels. The postoperative total leukocyte count of untreated recipients was higher than that of the other groups, but the difference was significant only on the 3rd and 4th posttransplant days. Animals treated with azathioprine 8 and 5-FU had significantly lower amylase concentration throughout the posttransplant period, and serum amylase values correlated well with the preservation of the normal architecture of the pancreatic tissue after transplantation. Pancreatitis and peritonitis were present in more than 1/2 the dogs treated with azathioprine alone, whereas there was no case of pancreatitis and a decreased incidence of peritonitis in animals given azathioprine plus 5-FU.

4741 PHOSPHOLIPASE A ACTIVITY IN EFFUSIONS FROM PATIENTS WITH ACUTE PANCREATITIS. *(Fr.)* Thouvenot, J. P.; Durand, S.; Douste-Blazy, L. (Purpan Hosp., Toulouse, France). *Arch. Fr. Mal. App. Dig.* 63(6):479-483, 1974.

By measuring the amount of ^{32}P-phosphatidyl ethanolamine hydrolyzed in the presence of sodium deoxycholate and the absence of trypsin, phospholipase A activities were determined in ascites fluid from five patients with acute pancreatitis and from seven patients with alcoholic cirrhosis and no evidence of pancreatic damage. Results are expressed in nM of ^{32}P-lysophosphatidyl ethanolamine (LPE) liberated by 1 ml of ascites fluid. After 5 min incubation, LPE values ranged from 70 to 815 nmol/ml (mean 330 nmol/ml) in fluid from patients with pancreatitis. The variation in these results is probably due to differences in the severity of the pancreatic lesion and the distance between the foci of inflammation and the peritoneum and does not appear to be caused by enzyme inactivation during the determination or by activation of prophospholipase in the ascites

fluid. In two patients, plasma phospholipase activities were lower than those in ascites fluid, suggesting that the enzyme does not originate in the circulatory system. Phospholipase A activities ranged from 25 to 159 nM LPE/ml/5 min (mean 58 nM/ml/5 min) in ascites fluid from cirrhotics). Individual variations in phospholipase A activities in cirrhotics might be due to the chronicity of effusion and absorption of fluid. Student's test demonstrated that for every incubation time employed (5-60 min), phospholipase A activities were significantly higher in ascites fluid from patients with acute pancreatitis than in that from cirrhotics. No lysophospholipase activity was detected in either group.

4742 EPIDEMIOLOGY AND AETIOLOGY OF CHRONIC CALCIFYING PANCREATITIS IN BELO HORIZONTE, BRAZIL. *(E.)* Dani, R.; Nogueira, C. E. D.; Furtado, M.; Leal, S. (Sch. Med., Fed. Univ. Minas Gerais, Belo Horizonte, Brazil). *Rend. Gastroenterol.* 6(3):153-156, 1974.

To determine the frequency and etiology of chronic calcifying pancreatitis (CCP) in Brazil, data from autopsy records and personal cases were compared with findings in healthy controls matched for race, sex, profession, and age. Among 6060 autopsies, diseases of the digestive system were the cause of death in 1205 cases. Of the latter, 256 cases showed pancreatic involvement which was diagnosed as chronic pancreatitis in 13. Thus, the disease occurred in 0.21% of the total cases and in 1.07% of diseases of the digestive system. Of 54 personal cases, 43 (41 men and 2 women) were alcoholic. The difference in age at onset of symptoms between this group and the 11 nonalcoholics (6 women and 5 men) was significant. Moreover, 54.5% of persons with CCP of unknown etiology were aged 6-25 yr, whereas only 1 alcoholic was under 25 yr. Comparison of dietary intake of 27 CCP patients and matched controls revealed that alcohol consumption and carbohydrate and calorie intake were significantly higher in patients than in controls. Protein intake (mainly vegetable proteins) was also significantly higher in the patients. These results do not support the possibility of protein deficiency being a cause of pancreatitis.

4743 CHANGES DUE TO PANCREATIC CYSTS SEEN ON RETROGRADE ENDOSCOPIC CHOLEDOCHO-PANCREATICOGRAMS. *(Ger.)* St. Stender, H.; Seifert, E.; Misaki, E.; Luska, G.; Lesch, P. (Med. Coll., Hannover, Germany). *Fortschr. Gen. Rontgenstr. Nuklearmed.* 120(3):253-258, 1974.

Endoscopic choledocho-pancreaticography (ERCP) was evaluated in the diagnosis of pancreatic disease. This method not only revealed changes in the pancreatic duct due to chronic inflammations and tumors but also demonstrated cysts directly or resultant secondary changes in the pancreatic and bile ducts. Among 612 ERCP's, 10 patients were found with pancreatic cysts. The following signs were characteristic: the cyst may be filled from the pancreatic duct; round void spaces in the contrast agent fillings showing the peripheral ducts and the parenchyma may be due to cysts; the pancreatic duct may be abruptly occluded or be of the tapering type; narrowing or displacement of the intrapancreatic portion of the common bile duct may result from cysts. Distinction between benign and malignant cystic changes in the pancreas is difficult. Following ERCP the patient must be observed carefully for complications due to possible activation of inflammatory processes.

4744 OBSERVATIONS ON ACUTE PANCREATITIS: A POST-MORTEM REVIEW. *(E.)* Gillespie, W. J. (R. Infirm. Edinburgh, Scotland). *J. R. Coll. Surg. Endinb.* 20(1):36-41, 1975.

Postmortem findings in 126 cases of acute pancreatitis (47 men and 79 women) revealed 53 cases of gallstones compared to an expected incidence of 29.95. The stones were in the gallbladder only in 30 cases, in the common bile duct in 16, and were obstructing the ampulla of Vater in 7. Pancreatic abscesses occurred in 14 patients and pseudocysts in 9; fat necrosis was visible to the naked eye in 108 cases. The association between acute pancreatitis and gallstone disease was highly significant and is hypothesized to be the result of the high incidence of gram-negative bacteria associated with cholelithiasis. The frequently focal nature of the pancreatitis is also compatible with an infective origin; in this series, for example, the site of pancreatic disease was in the head alone in 27 cases including 10 subjects with gallstones. Only 3 patients had a history of alcoholism. Despite the increasing accuracy of diagnosis, failure of diagnosis still occurs in acute pancreatitis. The majority of patients are elderly or have multiple preexisting disorders.

4745 MORPHOMETRIC-STATISTICAL ANALYSIS OF PANCREATIC ARTERIES IN CHRONIC PANCREATITIS. *(Ger.)* Kaiser, G.; Hommel, G. (Inst. Pathol., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Virchows Arch. [Pathol. Anat.]* 365(2):103-118, 1975.

Changes in pancreatic vessels in chronic pancreatitis and whether the diameter of the arterial vessels is reduced, are statistically examined. Histological preparations from two groups, treated with the van Gieson stain, were used for the investigation: Group I consisted of 50 subjects with healthy pancreatic tissue, and group II of 50 operative cases of chronic pancreatitis. At a 320-fold magnification, media thickness, vessel diameter, and intima thickness of four arterial vessels in each case were determined by a measuring scale in the lens of a light microscope. The test results were transferred to perforated cards and analyzed by computer. Arterial vessels in the endothelium in case of pancreatitis showed cell nuclei projecting into the lumen. Especially remarkable was the thickening of the subendothelial intima tissue in the form in intima-fibrosis and intima-elastosis; atrophy of the media was noted in later stages. The lumen of the arterial vessels seemed to be considerably constricted as compared to that of the venous vessels. The relations between media thickness and vessel radius in both groups were determined and, using regression lines as a model,

vessel constriction was found to be statistically significant. Although regression lines for the corresponding thickness were quite close, they were still significantly different. This remaining difference could have been caused by alteration in the media or also by additional influential characteristics that differ in the two test groups. The relation between vessel radius and media thickness was examined with regard to the following variables: distribution of age, sex, and phases of parenchyma reduction. Results show that the differences between the two groups were hardly influenced by age. The different distribution of sex seems to weaken the results more than to intensify it. Finally, the phase of parenchyma reduction corresponds to the extent of vessel alteration. These vessel alterations probably represent an adaption of the reduced parenchyma to the lowered demand for blood. On the other hand, it is rather unlikely that such alteration is an important pathogenetic factor of chronic pancreatitis or a consequence of exterior pressure caused by perifocal inflammation and incisive cicatricial tissue.

4746 THE DIAGNOSIS AND THERAPY OF ACUTE PANCREATITIS. *(Sl.)* Moravec, R.; Chorvat, V.; Bencur, J.; Babala, J.; Lesko, A.; Ohradka, B.; Labas, B. (Med. Fac., Comenius Univ., Bratislava, Czechoslovakia). *Rozhl. Chir.* 53(11):771-779, 1974.

4747 THIRTY-THREE CASES OF NECROTIZING PANCREATITIS TREATED BY PANCREATECTOMY. *(Fr.)* Guivarch, M.; Mouchet, A.; Marquand, J. (no affil.). *Chirurgie* 100(12):891-892, 1974.

4748 THE SURGICAL TREATMENT OF ACUTE PANCREATITIS. *(Rus.)* Shapkin, V. S.; Khorev, G. N.; Sotnichenko, B. A.; Nekhay, K. Kh. (Vladivostok Med. Inst., USSR). *Klin. Khir.* (4):51-56, 1975.

4749 EVALUATION OF SURGICAL METHODS FOR TREATING DESTRUCTIVE FORMS OF ACUTE PANCREATITIS. *(Rus.)* Moroz, I. M. (Lvov Med. Inst., USSR). *Khirurgiia (Mosk.)* 616(3):31-36, 1974.

4750 EXPERIENCE IN COMBINED TREATMENT OF ACUTE PANCREATITIS. *(Rus.)* Postlov, M. P. (Tashkent Med. Inst., USSR). *Khirurgiia (Mosk.)* 616(3): 26-31, 1974.

4751 TREATMENT OF ACUTE PANCREATITIS. *(Fr.)* Hollender, L. F. (Univ. Hosp. Cent., Strasbourg, France). *Lyon Chir.* 71(2):141-144, 1975.

4752 INTENSIVE CARE IN ACUTE PANCREATITIS. *(Ger.)* Pross, E. (Surg. Clin., Univ. Mainz, Germany). *Langerbecks Arch. Chir.* 337(Suppl.):251-257, 1974.

4753 CHANGES IN THE BLOOD CLOTING SYSTEM IN ACUTE PANCREATITIS. *(Rus.)* Moreino, M. S.; Molodenkov, M. N.; Efimov, V. S. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Khirurgiia (Mosk.)* 616(3):36-40, 1974.

4754 EXOCRINE SECRETORY FUNCTION OF THE PANCREAS AFTER ACUTE PANCREATITIS. *(Cz.)* Stastna, R.; Skala, I.; Skala, J.; Zvolankova, K. (Res. Cent. Metabol. Nutr., Prague, Czechoslovakia). *Cesk. Gastroenterol. Vyz.* 28(6):388-396, 1974.

4755 CONTENT OF HIGHER FATTY ACIDS AND TOTAL LIPIDS IN THE SERUM OF PATIENTS WITH ACUTE PANCREATITIS. *(Rus.)* Khomiakov, A. E.; Keda, B. I. (N.I. Pirogov 2nd Moscow Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11):70-79, 1974.

4756 INFLUENCE OF DIMEDROL AND ε-AMINOCAPROIC ACID ON THE LEVEL OF HISTAMINE AND SEROTONIN IN BLOOD FROM PATIENTS WITH ACUTE CHOLECYSTOPANCREATITIS. *(Rus.)* Likhachov, V. A.; Bhatta, A. D. (Kharkov Med. Inst., USSR). *Khirurgiia (Mosk.)* 616(3):62-66, 1974.

4757 ACUTE PANCREATITIS COMPLICATING IDIOPATHIC HYPERLIPEMIA. *(E.)* Gabrylelewicz, A.; Kinalska, I. (Med. Acad., Bialystok, Poland). *Mater. Med. Pol.* 6(3):220-222, 1974.

4758 ACUTE HAEMORRHAGIC PANCREATITIS FOLLOWING L-ASPARAGINASE THERAPY IN ACUTE LYMPHOBLASTIC LEUKAEMIA -- A CASE REPORT. *(E.)* Tan, C. L.; Chiang, S. P.; Wee, K. P. (Outram Road Gen. Hosp., Singapore). *Singapore Med. J.* 15(4):278-282, 1974.

4759 RESULTS FROM THE SURGICAL TREATMENT OF CHRONIC PANCREATITIS. *(Ger.)* Lankisch, P. G.; Fuchs, K.; Schmidt, H.; Creutzfeldt, W.; Peiper, H. J. (Med. Clin., Univ. Gottingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:552-554, 1974.

4760 THE USE OF RETROGRADE PANCREATOGRAPHY FOR ESTABLISHING INDICATIONS FOR SURGICAL TREATMENT OF CHRONIC PANCREATITIS. *(Ger.)* Manegold, B. C. (Fac. Clin. Med., Univ. Heidelberg, Mannheim, Germany). *Therapiewoche* 24(51):6048-6053, 1974.

4761 THERAPEUTIC METHODS FOR CHRONIC PANCREATITIS. *(Rus.)* Bondar, Z. A.; Tuzhilin, S. A. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(7):32-40, 1974.

4762 COMPENSATORY ABILITIES OF THE PANCREAS IN CHRONIC PANCREATITIS. *(Rus.)* Biagovidov, D. F.; Danilov, M. V.; Karvonidi, P. G.; Raben, N. A. (A. V. Vishnevskii Inst. Surg., Moscow, USSR). *Khirurgiia (Mosk.)* 616(9):62-67, 1974.

4763 CHRONIC RECURRENT PANCREATITIS IN STENOSIS OF THE CELIAC ARTERY. *(Ger.)* Rosellen, E.; Kremer, G. J.; Schnellbacher, E. (Med. Clin., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med* 80:550-551, 1974.

4764 MANAGEMENT OF MILD AND SEVERE ALCOHOLIC PANCREATITIS. *(E.)* Kowlessar, O. D. (Jefferson Med. Coll., Thomas Jefferson Univ., Philadelphia, Pa.). *Ann. NY Acad. Sci.* 252:208-213, 1975.

4765 ALCOHOLIC PANCREATITIS. CHRONIC CALCIFYING PANCREATITIS. *(Ger.)* Sarles, H.; Sahel, J. (Res. Unit Gastrointest. Dis., INSERM., Marseille, France). *Verh. Dtsch. Ges. Inn. Med.* 80: 1740-1745, 1974.

4766 ALCOHOLISM, ALCOHOLIC PANCREATITIS, AND PANCREATIC SECRETION. *(E.)* Dreiling, D. A. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Ann. NY Acad. Sci.* 252:187-199, 1975.

4767 THE EFFECT OF ALCOHOL ON THE EXOCRINE PANCREAS. *(Ger.)* Goebell, H.; Hotz, J. (Dep. Intern. Med., Univ. Ulm, Germany). *Therapiewoche* 24(51):6040-6046, 1974.

4768 PSEUDOCYST OF THE PANCREAS. EXPERIENCE WITH 70 CASES. *(E.)* Hollender, L. F.; Bur, F.; Marrie, D.; Alexiou, D.; Beaugie, J. M. (Univ. Hosp. Cent., Strasbourg, France). *J. R. Coll. Surg. Edinb.* 20(1):42-48, 1975.

4769 PANCREATIC PSEUDOCYSTS. *(Fr.)* Adloff, M. (Surg. Cent., Univ. Strasbourg, France). *Rev. Med. Liege* 30(1):1-6, 1975.

4770 THE PATHOGENESIS OF PANCREATIC CYSTS AND SURGICAL POLICY FOR THEIR TREATMENT. *(Rus.)* Seniutovich, V. F.; Shevchuk, M. G.; Genyk, S. N. (Ivano-Frankovsk Med. Inst., USSR). *Klin. Khir.* (4):44-46, 1975.

4771 OPERATIONS FOR TWO PANCREATIC PSEUDOCYSTS. *(Rus.)* Petrov, V. I.; Muriev, A. I.; Siniaev, V. P. (S. M. Kirov Mil. Med. Acad., Leningrad, USSR). *Klin. Khir.* (4):62-63, 1975.

4772 THE EFFECT OF OXYTETRACYCLINE ON EXOCRINE PANCREATIC FUNCTION. *(Ger.)* Fleischer, K.; (Med. Clin., Univ. Wurzburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:1543-1546, 1974.

4773 EXOCRINE PANCREAS FUNCTION IN OLD AGE. *(Ger.)* Schulz, H. J.; Dietze, F.; Bruschke, G. (German Gerontol. Res. Proj., Humboldt Univ., Berlin). *Dtsch. Gesundheitswes.* 29(45):2126-2129, 1974.

4774 PANCREATIC BIOPSY IN THE DIAGNOSIS OF PANCREATITIS. *(Ger.)* Bader, G. (Rostock-Sudstadt Dist. Hosp., Germany). *Dtsch. Gesundheitswes.* 29(45):2129-2134, 1974.

4775 CHRONIC FORMS OF PLEURISY WHICH ORIGINATE IN THE PANCREAS: THEIR SURGICAL TREATMENT. *(Fr.)* Marland, P.; Bersay, C.; Lacombe, M.; Beaufil, B. (Bichat Hosp., Paris, France). *Poumon Coeur* 30(3):199-206, 1974.

4776 THE RETROGASTRIC TRANSVENTRICULAR APPROACH FOR DRAINAGE OF THE "SUPRAGASTRIC" PANCREATIC PSEUDOCYST. *(E.)* Greenstein, A. J.; Dreiling, D. A. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 63(4):312-315, 1975.

4777 ACUTE PANCREATITIS. *(E.)* Trapnell, J. E. (R. Victoria Hosp., Boscombe, England). *Br. J. Hosp. Med.* 12(2):193-203, 1974.

4778 PATTERNS OF INCIDENCE IN ACUTE PANCREATITIS. *(E.)* Trapnell, J. E.; Duncan, E. H. L. (R. Victoria Hosp., Boscombe, England). *Br. Med. J.* 2(5964):179-183, 1975.

4779 PANCREATITIS AFTER RENAL TRANSPLANTATION. *(E.)* Corrodi, P.; Knoblauch, M.; Binswanger, U.; Scholzel, E.; Largiader, F. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Gut* 16(4):285-289, 1975.

See also, 3796, 3992, 4197, 4245, 4246, 4250, 4252, 4253, 4254, 4255, 4664, 4729, 5193.

LIVER AND BILIARY TRACT

4780 POST-JEJUNOILEAL-BYPASS HEPATIC DISEASE. ITS SIMILARITY TO ALCOHOLIC HEPATIC DISEASE. *(E.)* Peters, R. L.; Gay, T.; Reynolds, T. B. (Univ. South. Calif. Sch. Med., Los Angeles). *Am. J. Clin. Pathol.* 63(3):318-331, 1975.

Liver structural changes were followed by serial hepatic biopsies in five patients with hepatic disease following jejunal-bypass for extreme obesity. Three patients were nonalcoholics while two decreased their intake following surgery. Hepatic dysfunction became manifest 2.5-5 months after bypass when weight losses ranged from 65-127 lb. Nausea and vomiting, abdominal pain and hepatomegaly were present

in all five while ascites and splenomegaly were detected in three. Laboratory findings, which included hyperbilirubinemia, decreased albumin, prolonged prothrombin time, moderate elevation of alkaline phosphatase and transaminases with the rise in SGOT greater than SGPT, were identical to those seen in acute alcoholic hepatic disease. Hepatic microscopic changes included centrilobular or focal alcoholic hyalin deposition, intrasinusoidal collagenosis, fatty degeneration and neutrophilic infiltration. Four of the five patients died in hepatic failure despite hyperalimentation and re-establishment of intestinal continuity. It is concluded that further study of the mechanism of post-bypass hepatic disease may help in understanding the pathogenesis of alcoholic liver disease.

4781 ARTERIAL INFUSION CHEMOTHERAPY OF HEPATOMA. *(E.)* Cady, B.; Oberfield, R. A. (Lahey Clin., Boston, Mass.). *Lahey Clin. Found Bull.* 24 (2):49-55, 1975.

Regional arterial infusion chemotherapy for carcinoma of the liver was used in 18 of 54 patients. Of the remainder, five underwent resection, and the rest, untreated, served as non-concurrent controls. Tumors were graded as well-, moderately-, or poorly differentiated; 17 tumors were classified as liver cell carcinomas and one was classified as cholangiolar. Infusion catheters placed after exploration revealed that the extent of the disease precluded curative hepatic lobectomy. 5-Fluorodeoxyuridine was given in daily doses of 0.3 mg/kg. Occasionally, fluorouracil was used instead, at 3-5 mg/kg/day. Methotrexate was used in one patient (5 mg/kg/day). Treatment was continued as long as catheter and arterial patency could be demonstrated angiographically. Of the 18 patients given infusion, five died as a result. Of these, two died from fibrinolysin production with hemorrhage and pneumonia; one from hemorrhage of esophageal varices, and one from subphrenic abscess and pneumonia, all in the first yr of the program. In the last 5 yr, there was only one treatment related to death. Of 11 males (18-68 yr), median survival rate after elimination of three treatment-related deaths was 16.5 months. Of seven females (12-69 yr), median survival time after elimination of two treatment related deaths was 8 months. Excepting four patients who had no postoperative follow-up, median hepatic arterial patency time was 8.5 weeks. Of 18 patients, 11 were definitely improved, and eight were substantially improved, symptomatically. During the last 5 yr, 10 of 12 were improved, and eight of 12 were substantially improved. Symptomatic improvement was accompanied by a 16.5 month increase in median survival time. Between 1957 and 1962, two patients had resection and 21 no treatment; one survived over 1 yr. Between 1962 and 1965, six patients were given infusion and six no treatment; one survived over six months. After 1966, 12 patients were given infusion, three had resection, and eight had no treatment; 11 survived 6 months, and seven over 1 yr. The difference in survival time between patients not undergoing resection before 1962 and after 1966 is significant. No patient on infusion had all laboratory or clinical features return to normal. It is concluded that hepatic carcinoma may be substantially improved by infusion chemotherapy if resection is not possible.

4782 KINETICS OF RADIOCOPPER IN WILSON'S DISEASE. *(Ger.)* Willvonseder, R.; Goldstein, N. P.; Tauxe, W. N. (Mayo Clin. Mayo Found., Rochester, Minn.). *Wien. Z. Inn. Med.* 54(5/6):226-233, 1973.

Plasma incorporation, liver uptake, urinary and fecal excretion, and body retention of i.v. administered ^{64}Cu and ^{67}Cu were examined in 35 homozygous and heterozygous carriers of Wilson's disease, and in a normal control group. Plasma incorporation in heterozygous patients was lower than in normal subjects, and there was practically no incorporation at all in homozygous patients with manifest Wilson's disease. The liver uptake was slightly higher in heterozygous patients than in normal subjects, but it showed steady increase in homozygous patients, and reached extremely high values in the preclinical stages of Wilson's disease. The urinary and fecal excretion was lower in heterozygous patients than in the control group, and the lowest values were found in preclinical stages. The urinary excretion was higher in untreated homozygous patients with manifest Wilson's disease than in heterozygous patients and in the control group, while the fecal excretion was practically zero in the former group. The mean biological half-life period of ^{64}Cu was determined to be 17 days in the control group, 39.4 days in heterozygous patients, and 97 days in homozygous untreated patients. The results indicate that copper is not bound to ceruloplasmin in patients with Wilson's disease. The liver copper uptake mechanism is still compensated in preclinical stages, but it is decompensated in clinical stages of Wilson's disease. Due to their high discriminatory efficiency, radiocopper investigations are recommended in all patients suspected of an error in copper metabolism.

4783 PRIMARY ANGIOSARCOMA OF THE LIVER. REVIEW ARTICLE. *(E.)* Alrenga, D. P. (Hektoen Inst. Med. Res., Chicago, Ill.). *Int. Surg.* 60(4): 198-203, 1975.

The characteristics of primary angiosarcoma of the liver are reviewed for both the adult and infantile forms. The disease formed 1.8% of all primary liver cancers in a review of the literature between 1954 and 1974, and had an autopsy incidence of 6/100,000. There was no geographic variation in incidence. The most common etiologic agent was thorium dioxide, responsible for at least 50 cases since 1947. Angiosarcoma comprised over 1/3 of all thorium dioxide-induced malignancies: eight cases of angiosarcoma in 28 cases of postnecrotic necrosis induced by arsenic trioxide; one caused by γ-radiation from a radium needle lodged in the heart; and one in a patient treated for 17 yr with arsenic for psoriasis. There were about 20 deaths attributed to vinyl chloride-induced angiosarcoma. Clinically, 15% of the patients were under 40 yr of age, and males predominated 2:1 over females. The commonest symptoms were rapid hepatic enlargement with epigastric pain, ascites (often hemorrhagic), and jaundice, although the mode of presentation was quite variable in general. Infantile

hemangioendothelioma occurred in children under 4 yr of age; 70% were diagnosed before 6 months of age. Hepatomegaly was the only consistent finding on physical examination. Abdominal distension, vomiting, diarrhea, or constipation were the most common symptoms. Gross morphology of both angiosarcoma and infantile hemangiendothelioma were very similar. Tumors were solitary, multicentric, or diffuse, and varied from fleshy nodular masses to large blood filled cycts. Histopathologically, angiosarcoma, as opposed to the infantile form, had more nuclear atypia, mitotic activity, and giant cell formation. The most distinguishing feature was the presence of intercommunicating spaces lined by atypical endothelial cells confined within a thin reticulin network. Unique microscopic features also included neoplastic cells simulating Kupffer cells, extramedullary hemopoeisis within the tumor's vascular spaces, and ability of the tumor cells to ingest hemosiderin. The histologic pattern of the infantile form showed either vascular spaces lined by plump endothelial cells and separated by collagenous and myxoid stroma, or poorly formed vascular spaces with large pleomorphic endothelial cells and extramedullary hemopoeisis. Adult angiosarcoma was commonly associated with fibrosis or cirrhosis and the infantile forms with cutaneous hemangioma. Angiosarcoma had an unfavorable prognosis, while the infantile form had a more favorable one, with a 50% survival rate.

4784 EXCHANGE TRANSFUSION FOR REYE'S SYNDROME. *(E.)* Lewis, J. D.; Varma, R. R.; Harrington, G.; Caspar, J.; Ackamnn, J. J.; Darin, J. C. (Med. Coll. Wisconsin, Milwaukee). *J. Surg. Res.* 18(3):327-329, 1975.

Patients with Reye's syndrome, all in grade IV-V coma, were treated by exchange transfusion. Blood components were used for more efficient use of resources. Packed blood cells were washed with saline and combined with fresh-frozen citrate-phosphate-dextrose plasma. Cerebral edema was treated by administration of dexamethasone and mannitol, and the colon was "sterilized" with neomycin p.o. and *via* enema and fructose *via* enema. A total of 21 exchanges were performed on the nine patients. One patient responsed rapidly to one exchange. Six required two exchanges; five of these responded. Two required four exchanges before response. One patient was given peritoneal dialysis. Computerization of EEG activity showed significant diminution of delta activity with associated increase in theta and alpha activity in those treated by exchange transfusion, but not in the patient undergoing dialysis, until exchange transfusion had reversed the clinical course. It is concluded that exchange transfusion is extremely effective in the treatment of profound Reye's syndrome.

4785 'PURE' HEPATIC FORMS OF WILSON'S DISEASE IN CHILDREN: TEN OBSERVATIONS. *(Fr.)* Odievre, M.; Vedrenne, J.; Landrieu, P.; Alagille, D. (Parrot Hosp., Bicetre, France). *Arch. Fr. Pediatr.* 31(2/3):215-222, 1974.

Observations of 10 cases of Wilson's disease in children are presented. In all cases, the involvement was predominantly or exclusively hepatic and included cirrhosis (nine cases), intrahepatic portal hypertension (three), chronic active hepatitis (nine), and acute anemia and hepatocytic insufficiency due to acute transitory hemolysis (five cases, fatal in four). In one case, Wilson's disease was diagnosed by systematic examination following the deaths of two siblings from acute hemolysis and hepatocytic insufficiency caused by Wilson's disease. The clinical and biological polymorphism of Wilson's disease makes diagnosis difficult. The authors suggest that certain features of hepatic involvement are indicative of a metabolic origin: parallel decrease in the different factors of the prothrombic complex; coexistence of hepatic symptoms with signs of renal tubular involvement; association of hepatic portal fibrosis with steatosis; hepatic cytolysis; and normal levels of α_2-globulins associated with other hepatic symptoms. Definitive diagnosis is demonstrated by ceruloplasmin concentration below 200 mg/liter, plasma copper concentration below 60 µg/100 ml, and cupruria greater than 100 µg/24 hr. It is concluded that an elevated concentration of hepatic copper resolves diagnosis in doubtful cases.

4786 SPECTRUM OF LIVER DISEASE IN RENAL TRANSPLANT RECIPIENTS. *(E.)* Ware, A. J.; Luby, J. P.; Eigenbrodt, E. H.; Long, D. L.; Hull, A. R. (Univ. Texas Southwest. Med. Sch., Dallas, Texas). *Gastroenterology* 68(4):755-764, 1975.

Renal transplant patients over a 4 yr period were reviewed with an attempt to define episodes of liver disease in these patients in terms of their nature, etiology and prognosis. There were 82 patients at risk in development of liver disease. They were observed for 6 months to 4 yr, during which time they were frequently assessed for hepatic dysfunction clinically and by laboratory tests including serum bilirubin, serum alkaline phosphatase, and SGOT. Determination of the hepatitis B surface antigen (HB_sAg) were performed monthly, and at least one serum sample from each patient was assayed by solid phase radioimmunoassay, and cytomegalovirus (CMV) antibody was assayed by complement fixation. CMV was also assayed by innoculation of cultured cells with samples of urine or homogenized liver. The minimum criteria for liver disease were two consecutive elevated SGOT levels, 1 week apart. Of the 82 patients, 31 (38%) developed evidence of liver disease after the transplant. Of these, eight cases were acute, self limited, reversible, and benign; two were acute, and fatal hepatitis, and 21 were a form of chronic disease with cirrhotic potential. The development of the disease was not influenced by age, sex, nature of the kidney disorder, source of donor, quality of HLA tissue match, or the dosage of azathioprine and steroid therapy given to all patients. Of the 31 cases, 25 began within 6 months of transplantation. The eight cases of reversible disease lasted from 1-20 weeks. A biopsy performed in one case showed multiple fresh granulomata. Typical CMV inclusions were found in enlarged and degenerating hepatocytes. HB Ag was not present in any case. In six of the cases, there was seroconversion to CMV within a few

weeks of the disease's onset, and CMV was found in the urine in three cases. Of the two patients with acute fatal disease, one was HB_sAg positive. The other was negative, but CMV seroconversion coincided with the onset of the illness. Of the 21 patients with chronic disease, 16 underwent seroconversion to CMV. Follow-ups of from 6-40 months showed constant liver function test results. In three patients, there were five episodes of acute reversible icterus, probably etiologically unrelated. Three of the 21 patients had persistent HB_sAg antigenimia. One of these, however, was positive for HB_sAg for 2 1/2 yr before any abnormality of liver function was detected. It is suggested that CMV was a likely cause for a majority of the cases, while hepatitis B may have been implicated in a few, but the results are not conclusive.

4787 INVESTIGATION OF THE HEPATIC CATALASE ACTIVITY IN PATIENTS WITH MALIGNANT TUMORS AND FATTY LIVER. *(Ger.)* Guelzow, M.; Falkenhagen, D.; Huetter, J.; Arendt, R.; Teichmann, W. (Fac. Med., Univ. Rostock, Germany). *Z. Inn. Med.* 29(23): 959-964, 1974.

The liver catalase activity was determined by manometry in biopsy specimens from 32 normal subjects, 31 patients with malignant tumors of different localizations, and in 25 patients with fatty liver to establish the relation between the catalase activity and the pathological state. The liver catalase activity was highest with 13.29 IU/mg in normal subjects, and significantly lower in tumor patients, and especially in patients with moderate and serious fatty liver. The catalase activity was lowest in patients with serious degree of fatty liver, being significantly lower than in patients with moderate degree of fatty liver. The catalase activity, while not age-dependent, was highest in the 51-70 yr age group. No significant sex-conditioned difference was established. The difference between tumor patients with and without metastases was insignificant.

4788 HEPATIC ARTERY LIGATION AND CYTOTOXIC INFUSION IN TREATMENT OF LIVER NEOPLASMS. *(E.)* Zike, W. L.; Safaie-Shirazi, S.; Gulesserian, H. P. (Univ. Iowa Hosp., Iowa City). *Arch. Surg.* 110(5):641-643, 1975.

Hepatic artery ligation with distal infusion of the artery with cytotoxic drugs was used to treat 38 patients with primary or secondary hepatic neoplasms. None showed evidence of major extrahepatic metastases. Laparotomy was performed, the primary tumor was resected whenever possible, and the hepatic artery ligated distal to its extrahepatic branches. A catheter was inserted distal to the ligation and was ligated to the artery at two or more sites. The lesser omentum, falciform ligament, and triangular ligament were transected and the gallbladder removed. The catheter was kept open by constant infusion of saline and heparin solution, and when liver enzymes returned to preoperative levels, patients were infused with 12 mg/kg/day fluorouracil in heparinized saline. Treatment was discontinued when the catheter became displaced, became clogged by clotting, or when drug toxicity appeared. The average fluorouracil infusion lasted 45 days. The median survival time was 13.3 months; the longest survival was up to 44 months. Of the eight surviving patients, median survival time was 17 months. The primary tumor was in the large bowel in 25 patients, and in the liver in five. Of the 38, three had ligation only, with no infusion. There were marked postoperative elevations in alkaline phosphatase, SGOT, and LDH. Values returned to normal within 8 days. It is concluded that this method does not destroy the entire tumor, but does result in substantial palliation with minimal morbidity and mortality.

4789 THE DIAGNOSIS OF JAUNDICE BY THE MINI-LAPAROTOMY OPEN TRANSHEPATIC CHOLANGIOGRAM. *(E.)* Stein, H. D. (Lincoln Hosp., Bronx, N.Y.). *Ann. Surg.* 181(4):386-389, 1975.

Diagnosis of the etiology of obstructive jaundice was made by open transhepatic cholangiography (minilaparotomy) in 23 patients. Routine diagnostic procedures had failed in all. Five had ascites and four had elevated prothrombin levels. The liver was visualized through a 4 cm subxiphoid incision using local anesthesia. The catheter was inserted and contrast material injected into a bile duct under fluoroscopic control. Cholangiograms were attempted in all cases. Liver biopsies and cultures of bile and ascitic fluid were obtained when necessary. The procedure resulted in successful diagnosis in 21 of the 23 patients. In one unsuccessful case the cholangiogram was unsuccessful and surgery resulted in a diagnosis of intrahepatic cholestasis secondary to Gold therapy. In the other case, cholangiography showed obstruction of the common hepatic duct, but surgery disclosed carcinoma of the pancreas. Cholangiography was unsuccessful in two other patients in whom the procedure was attempted only perfunctorily because diagnosis was already evident by direct observation and biopsy. Of eight patients in whom non-surgical disease was diagnosed or diagnosis was in question, five had cholelithiasis. In the other three, precise diagnosis was changed but therapy was not. In 14 cases with high probability of surgical disease, three were saved surgery and clinical diagnosis was proved incorrect in four. There were no wound complications, bile leaks or hemorrhage, and future surgery was not interfered with in any patient. It is concluded that minilaparotomy is safe and reliable, and has a nearly 100% accuracy, low complication rate, and minimal limitations of use.

4790 HEPATIC CELL ADENOMAS, SPONTANEOUS LIVER RUPTURE, AND ORAL CONTRACEPTIVES. *(E.)* Ameriks, J. A.; Thompson, N. W.; Frey, C. F.; Appelman, H. D.; Walter, J. F. (Univ. Michigan Med. Cent., Ann Arbor). *Arch. Surg.* 110(5):548-557, 1975.

The clinical and pathological features of hepatic cell adenoma were studied in eight patients. All were women, aged 25-44 yr; all had been taking oral contaceptives containing a progestogen and estrogen combination, from 6 months to 9 yr, and all but

one had been at least twice pregnant before starting the drug. Average tumor size was 15 cm in diameter, but in three cases where the tumors ruptured, they were 18 cm in diameter. There was no correlation found between tumor size, clinical presentation, age, or duration of drug use. The three patients with ruptured tumors were treated by anatomical right lobectomy, and despite complications in each case, all survived and were followed up for 8 months to 3 yr. Two patients had expanding neoplasms which were excised with a rim of normal tissue. Both recovered with no significant morbidity. Three patients had asymptomatic neoplasms. Two of these were treated by excision with a rim of normal tissue, while the third was given an anatomical right lobectomy. All were well at follow-ups of 5 months, 3 yr, and 5 yr. All tumors consisted of well differentiated hepatocytes organized into sheets and cords, but without lobules, and were partly encapsulated by an incomplete fibrous membrane. It is suggested that there may be a relation between these tumors and oral contraceptives.

4791 LIVER IMPAIRMENT DURING CHRONIC HEMODIALYSIS AND AFTER RENAL TRANSPLANTATION. *(E.)* Nielsen, V.; Clausen, E.; Ranek, L. (Rigshosp., Copenhagen, Dnemark). *Acta Med. Scand.* 197(3):229-234, 1975.

The possible causes of aminotransferase elevation were elevated in patients treated with chronic hemodialysis and renal transplantation. Patients were treated with hemodialysis alone (23), hemodialysis and renal transplantation (37), or allotransplantation alone (19). Of the last, 12 had had previous hemodialysis treatment. Tests of serum alanine aminotransferase, prothrombin-proconvertin, and serum protein electrophoresis were done at least four times/month. Clinically, biochemically, and histologically, the patients were divided into five groups: 1) no clinical or biochemical evidence of liver disease; 2) elevated alanine aminotransferases, but no clinical evidence of disease; 3) anicteric hepatitis; 4) icteric hepatitis; and 5) elevated alanine aminotransferases possibly not due to viral hepatitis. Of the 60 patients given hemodialysis, 29 showed no liver disease, nine had transferase elevation, five had anicteric hepatitis, and 10 had icteric hepatitis. A final group consisted of seven patients in whom elevated alanine aminotransferase values were assumed to be due to bacterial sepsis in two, crush syndrome with anuria in two, iron contamination of dialysis water in one, cardiac arrest in one, and severe hemosiderosis of the liver in one. There was no significant rise in cytomegalovirus (CMV) titers in any. Of the 56 patients given renal allotransplants, 28 showed no liver impairment, nine had asymptomatic transferase elevation, 10 had anicteric hepatitis and four had icteric hepatitis. A final group consisted of five patients in whom elevated transferase values were assumed to be due to polycystic kidneys and liver cysts in one, urinary leakage and urinary tract infection in three, and hemosiderosis of the liver in one. CMV titers did not rise significantly in any. In all, 50% of the hemodialysis patients and 50% of the renal transplant patients had elevated transferase levels for longer or shorter periods, while the mortality from hepatitis was 13%. It is concluded that alanine aminotransferase elevation is not a contraindication for performing renal transplant.

4792 INFECTIOUS HEPATIC GRANULOMATOSIS: CLINICAL ASPECTS AND LIVER FUNCTION TESTS. *(Sp.)* Hernandez Guio, C.; Rey, R.; Aguirre, M.; Dominguez, A.; Patino, J. (Jimenez Diaz Found., Madrid, Spain). *Rev. Clin. Esp.* 133(1):39-44, 1974.

Of 33 patients with hepatic granulomatosis associated with infectious disease, 12 had tuberculosis, six brucellosis, and 15 leprosy. In four patients with tuberculosis, the liver was the only organ involved; cirrhosis was present in three patients with tuberculosis. Specific granulomatous lesions were found in all 28 patients from whom liver biopsies were taken. In general, symptoms were suggestive of granulomatous hepatitis. Fevers were present in all 12 patients with tuberculosis, in five of those with brucellosis, and in one with leprosy; seven other patients with leprosy ran fevers intermittently. Jaundice was present in seven of those with tuberculosis, in one with brucellosis and, intermittently, in seven with leprosy. Hepatomegaly was observed in 10 patients with tuberculosis, six with brucellosis, and 13 with leprosy. Splenomegaly was present in half of those with leprosy and tuberculosis and in four with brucellosis. Of the liver function tests performed, serum protein fractions exhibited the greatest changes in all three groups of patients. The most specific changes were noted in leprosy, probably as a result of immunological disorders. Turbidity tests were positive, but are considered nonspecific. SGOT values were pathological in four patients with tuberculosis and three with leprosy, suggesting the presence of parenchymal damage. However, none of these patients had histological evidence of cirrhosis. Serum alkaline phosphatase and BSP retention values are considered useful indicators of liver function in afebrile and nonjaundiced patients, resp. However, liver biopsies were the most valuable diagnostic tool. Although leprosy had been diagnosed in 14 patients before liver biopsies were taken, the etiology of hepatic granulomatosis was established only by liver biopsy in almost half of these patients.

4793 THE EFFECTS OF AGE AND LIVER DISEASE ON THE DISPOSITION AND ELIMINATION OF DIAZEPAM IN ADULT MAN. *(E.)* Klotz, U.; Avant, G. R.; Hoyumpa, A.; Schenker, S.; Wilkinson, G. R. (Vanderbilt Univ. Sch. Med., Nashville, Tenn.). *J. Clin. Invest.* 55(2):347-359, 1975.

The disposition and elimination of diazepam were studied in 21 patients with liver disease and in 33 healthy adults aged 15-82 yr. In healthy subjects, plasma diazepam concentration, as determined by gas chromatography, declined biexponentially with a clearance value of 20-32 ml/min following p.o. (10 mg) or i.v. (0.1 mg/kg) administration. Although clearance was not age-dependent, analysis of the i.v. data according to a two-compartment open model indicated that both the initial distri-

bution space and the steady state volume distribution of diazepam increased linearly with age. The increase in the steady state volume distribution was secondary to a change in the initial distribution space. Plasma half-life of desmethyldiazepam, the metabolite of diazepam, showed a similar age dependence although its peak concentration was lower and occurred later than the precursor. Both compounds bound extensively (97%) to plasma proteins. Patients with cirrhosis had a two-fold longer diazepan half-life (105.6 hr) than age-matched controls (46.6 hr) due primarily to similar decrease in total plasma clearance. Appearance of plasma desmethyldiazepam was delayed 3-8 hr compared to controls. Plasma diazepam clearance was also prolonged about two-fold in patients with acute viral and chronic active hepatitis. In cirrhotic and viral hepatitic patients, no correlation was found between plasma half-life or clearance and any of the routine liver function tests. It is concluded that: 1) diazepam plasma concentrations will not accumulate to a greater extent in older than in younger patients, thus making dosage modifications unnecessary; and 2) reduced drug clearance dictates the use of caution in prescribing diazepam to patients with acute or chronic liver disease.

4794 A STAGING SYSTEM FOR HEPATOCELLULAR CARCINOMA: PROGNOSTIC FACTORS IN UGANDIAN PATIENTS. *(E.)* Primack, A.; Vogel, C. L.; Kyalwazi, S. K.; Ziegler, J. L.; Simon, R.; Anthony, P. P. (Uganda Cancer Inst., Kampala). *Cancer* 35(5):1357-1364, 1975.

A functional-anatomical staging system for hepatocellular carcinoma was studied for its prognostic significance. Historical, clinical, and laboratory aspects of the scheme were examined in 72 untreated patients with hepatocellular carcinoma. The median survival for the group was 1 month. The presence of a serum bilirubin concentration of greater than 2 mg/100 ml or weight loss greater than 25% of body weight were the poorest prognostic features. Other factors having prognostic significance were visible abdominal collateral circulation, ascites, tumor differentiation, and elevated serum levels of alkaline phosphatase, SGOT, α-fetoprotein, and proline hydroxylase. Analysis of prognostic factors suggested a modification and simplification of the staging classification devised at an International Symposium on Liver Cancer in Kampala; three prognostically different groups of Ugandan patients were defined. The authors suggest that the staging scheme presented serve as a frame of reference for comparison of prognostic features in different populations of patients with hepatocellular carcinoma.

4795 HISTOLOGICAL CHANGES IN LIVER BIOPSIES OBTAINED AT THE TIME OF INTESTINAL BYPASS PROCEDURES FOR MORBID OBESITY. *(E.)* Krantz, B. E.; Schenken, J. R.; Swenson, S. A.; Vickstrom, D.; Pumphrey, H. (Univ. Nebraska Sch. Med., Omaha). *Nebr. Med. J.* 59(9):347-350, 1974.

Liver biopsies were performed in 46 patients undergoing jejunocolic bypass and on 107 patients undergoing jejunoileal bypass for morbid obesity (203-470 lb) or for complications of previous bypass procedures. Of 112 biopsies obtained at the time of primary bypass, 76% showed some degree of fatty change. Of 38 having a secondary bypass, 84% showed fatty liver. The majority of biopsies also had evidence of chronic portal or parenchymal inflammation, usually mild. Focal acute hepatitis, epithelioid granulomas and periportal fibrosis were evident in a few cases. In general, these changes were more commonly seen in patients following jejunocolic bypass. Histologic changes had usually progressed in 17 patients from whom liver biopsies had been obtained at the time of primary bypass and again during a second procedure 5 to 51 months later. It is concluded that liver biopsy should constitute an essential part of the prebypass evaluation of patients with morbid obesity.

4796 IMMUNOFLUORESCENT AUTOANTIBODY STUDIES IN THE DIAGNOSIS OF CHRONIC LIVER DISORDERS. *(E.)* Doniach, D. (Middlesex Hosp. Med. Sch., London, England). *Rend. Gastroenterol.* 6(3):192-203, 1974.

Serum antibodies detected by immunofluorescence are reviewed in relation to differential diagnosis in chronic liver disease. The incidence of antinuclear antibodies (ANA) is highest in lupoid hepatitis, but the ANA test is positive in 70-80% of patients with chronic active hepatitis (CAH) when Australia antigen-positive cases are excluded. The staining pattern is usually diffuse in CAH as opposed to speckled or nucleolar in primary biliary cirrhosis (PBC) and cryptogenic cirrhosis. Smooth muscle-related antibodies (SMA) are specific for CAH only if titers are greater than 80. Mitochondrial antibodies (AMA) are detected in 84-96% of patients with PBC; titers are high (200-6000) in about 1/2 the patients and moderate or low in the remaining. Liver/kidney microsomal antibodies (LKM) show a striking correlation with a form of CAH: the patients tend to be young and many progress to cirrhosis. Clinical and serological features are shown for six subgroups of CAH, the largest of which shows none of the serological markers. The other groups are Australia antigen-related (virus B), drug-induced, lupoid, PBC-related, and LKM-associated. Patients with lupoid hepatitis are mostly female and have SMA in addition to high titer ANA. The PBC-related cases are seen in much older women. Striking features of the LKM-associated cases are the lower frequency of ANA and SMA, the high proportion of males, and the defined icteric onset. All forms of acute hepatitis are associated with a high incidence of SMA, which disappear on recovery; ANA, AMA, and LKM reactions are rare and/or transient. Australia antigen tests are usually negative in lupoid and PBC-related CAH. Post-operative jaundice associated with halothane anesthesia is associated with a 100-fold increase in LKM. It is concluded that the serum antibodies detected in liver diseases are probably not responsible for destruction of hepatocytes but can be considered markers of other underlying pathogenic processes. Their constant presence only in certain types of disease suggests that they are not an epiphenomenon.

4797 SUBSTRATE INCORPORATION INTO HEPATIC LIPIDS AND PROTEINS IN VITRO IN PATIENTS WITH PRE-β HYPERLIPOPROTEINEMIA. *(E.)* Stakeberg, H.; Schersten, T. (Sahlgrenska Hosp., Univ. Goteberg, Sweden). *Acta Med. Scand.* 197(3):217-223, 1975.

The role of the liver in pre-β-hyperlipoproteinemia was studied in 13 patients with types IIB and IV of the disease who presented for surgery with uncomplicated gallbaldder disease, (and in one, atherosclerotic abdominal aortic disease). Of these, five were fed a sucrose-enriched diet for 14 days prior to surgery. There were 40 controls, all presenting for surgery with uncomplicated gallbladder disease, and having no other known disorder. Of these, four were fed a sucrose-enriched diet for 14 days. No patients had known alcoholic problems or were on oral contraceptives. All fasted from 8 pm the day before until surgery at about 8 am. Liver biopsy was performed immediately and the liver slices prepared and incubated for determination of rates of glycerol and fructose incorporation into liver lipids and glycogen, determination of leucine incorporation into proteins, determinations of lipid phosphorus, glycogen, and protein, and determination of the rate at which labeled precursors were incorporated into triglycerides, phosphoglycerides, the fatty acid moeity of glycerides, and glycogen. Patients with the disease and on an ordinary diet had phosphoglyceride, glycogen, and protein concentrations similar to control values. The five fed sucrose showed a high triglyceride concentration (55 μM/g), low phosphoglyceride concentration (19.7 μM/g), high glycogen concentration (7.2 mg/100 mg) and normal protein concentration. Phosphoglyceride concentration in liver from patients with the disease on ordinary diet was the same as the control value. In diseased patients not given sucrose, glycerol was incorporated in triglyceride at a higher rate than that of controls. There was also a trend toward increased incorporation rate of this substrate into the fatty acid glyceride moiety. In the sucrose-fed diseased patients, glycerol was incorporated into triglyceride at the same rate as those on the ordinary diet (254 μM/g). However, incorporation of glycerol and fructose (51.0, 17.4, resp.) into hepatic phosphoglycerides were lower after sucrose feeding in diseased patients than those on ordinary diet. There was also a lower glycerol incorporation rate into glyceride fatty acid moieties in sucrose-fed patients (1.9 μM/g). Controls fed sucrose had a higher leucine incorporation rate into hepatic proteins than controls on ordinary diets. It is concluded that the vulnerability of hepatic phosphoglyceride and protein metabolism to sucrose is increased in patients with pre-β-hyperlipoproteinemia.

4798 ANTIBIOTICS AND HEPATOBILIARY DISEASE. *(Fr.)* Metreau, J. M.; Berthelot, P. (Henri Mondor Hosp., Creteil, France). *Therapie* 29(3):369-375, 1974.

4799 CHANGES IN SINUSOIDS IN HEPATIC DISEASES. *(Rus.)* Kartashova, O. Ia.; Zaltsmane, V. K.; Zarinia, L. A.; Golubov, I. S. (Riga Med. Inst., USSR). *Arkh. Patol.* 36(8):40-46, 1974.

4800 THE ROLE OF ORNICETIL IN HEPATOLOGY. *(Gr.)* Dorf, G. (Hotel Dieu, Paris, France). *Galenus* 15(4):552-559, 1973.

4801 INVESTIGATION OF IMMUNOGLOBULINS IN CHRONIC LIVER DISEASES. *(Rus.)* Minogina, N. A.; Volkoslavskia, N. V. N. (Ukr. Inst. Postgrad. Med., Kharkov, USSR). *Vrach. Delo* (12):82-85, 1974.

4802 IMMUNOGLOBULINS IN CHRONIC LIVER DISEASES. *(Pol.)* Pokora, J.; Tomaszewski, J.; Daniluk, J.; Kozak, J.; Czarnecki, J. (Acad. Med., Lublin, Poland). *Pol. Tyg. Lek.* 29(43):1827-1829, 1974.

4803 CHANGES IN THROMBOCYTE FUNCTION IN LIVER DISEASE. *(Ger.)* Schulz, K. (Dep. Med., Univ. Rostock, Germany). *Munch. Med. Wochenschr.* 116(47):2067-2072, 1974.

4804 THE MANAGEMENT OF BLEEDING IN LIVER DISEASE. *(E.)* Parbhoo, S. (Frenchay Hosp., Bristol, England). *Br. J. Hosp. Med.* 13(1):17-28, 1975.

4805 PRESCRIBING IN PATIENTS WITH LIVER DISEASE. *(E.)* James, I. (R. Free Hosp., London, England). *Br. J. Hosp. Med.* 13(1):67-70, 73-76, 1975.

4806 THE EFFECT OF CHOLERETICS ON BILE FLOW IN LIVER AND BILIARY DISEASE. *(Ger.)* Bode, J. C.; Strohmeyer, G. (Med. Clin. II, Univ. Dusseldorf, Germany). *Leber Magen Darm* 4(2):65-71, 1974.

4807 ENCEPHALOPATHY IN LIVER DISEASE. *(E.)* Lunzer, M. (R. Free Hosp., London, England). *Br. J. Hosp. Med.* 13(1):33-44, 1975.

4808 COMBINED LAPAROSCOPY IN DISEASES OF HEPATOBILIARY SYSTEM. *(Rus.)* Dolgor, P.; Zhigzhidsuren, L. (State Med. Inst., Ulan Bator, Mongolian Peoples Republic). *Khirurgiia (Mosk.)* (1):80-82, 1974.

4809 DYNAMIC LIVER SCANS IN DIAGNOSIS OF LIVER AND BILIARY DISEASES. *(Rus.)* Makarenko, T. P.; Nasatkin, Iu. M.; Khutiev, Ts. S.; Mironov, S. P.; Upyrev, A. V. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Khirurgiia (Mosk.)* (11):74-78, 1973.

4810 COMBINED INSTRUMENTAL DIAGNOSIS OF LOCALIZED LIVER DISEASES. *(Rus.)* Epifanov, N. S.; Zhuravlev, V. A.; Korepanov, V. I. (Kirov Res. Inst. Blood Transfus., USSR). *Khirurgiia (Mosk.)* (11):68-74, 1973.

4811 NORMAL HEPATOGRAM IN THE NEWBORN. *(Sp.)* Torres Vera, M. T.; Villasmil Salas, J. A.; Ruiz Maury, A.; Diaz Isea, A. (Univ. Hosp., Maracaibo, Venezuela). *Rev. Fac. Med. Maracaibo* 6(1-4): 256-266, 1973.

4812 SERUM ALCOHOL DEHYDROGENASE IN LIVER DISEASE: MULTIVARIANT APPROACH TO INTERPRETATION OF CLINICAL AND PATHOPHYSIOLOGICAL FINDINGS. *(It.)* Di Somone, A.; Oriani, G. A.; Riegler, G.; Coltorti, M. (1st Fac. Med. Surg., Univ. Naples, Italy). *Fegato* 19(3):557-569, 1973.

4813 RADIOIMMUNOLOGICAL DETECTION OF AUSTRALIA ANTIGEN (HG Ag) IN SALIVA IN VARIOUS LIVER DISEASES. *(Ger.)* Broderson, M.; Stegmann, S. (Luitpold Hosp., Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:465-467, 1974.

4814 THE CORRELATION BETWEEN α_1-FETOPROTEIN (AFP) AND AUSTRALIA ANTIGEN (HB Ag) IN HEPATIC PATHOLOGY. *(It.)* Migneco, G.; Mansueto, S.; Tripi, S.; Didato, A. (Inst. Gen. Clin. Med., Med. Ther., Univ. Palermo, Italy). *Boll. Ist. Sieroter. Milan* 53(1):75-78, 1974.

4815 ALFA-FETOPROTEIN IN LIVER DISEASES IN TAIWAN. *(Ch.)* Sung, J. L.; Liaw, Y. F.; Lin, W. S. J.; Chen, T. H. (Coll. Med., Natl. Taiwan Univ., Taipei). *J. Formosan Med. Assoc.* 73(3): 178-183, 1974.

4816 STUDIES OF LIVER FUNCTION BY ^{131}I-LABELED BENGAL ROSE IN PATIENTS WITH MYOCARDIAL INFARCTION. *(Rus.)* Leschinsky, L. A.; Trusov, V. V.; Shemyakin, G. I. (Izhevsk Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11):79-85, 1974.

4817 HEPATIC LYMPHATICS DEMONSTRATED BY PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. *(E.)* Goldberg, H. I.; Dodds, W. J.; Lawson, T. L.; Stewart, E. T.; Moss, A. A. (Univ. California Sch. Med., San Francisco). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):415-419, 1975.

4818 PRIMARY RETICULOSARCOMA OF THE LIVER. *(It.)* Rosa, G.; Farello, G. A.; Chiamenti, C. (Inst. Spec. Surg. Pathol. Clin. Propedeut., Univ. Padua, Italy). *Acta Chir. Ital.* 30(2):193-201, 1974.

4819 CURRENT MANAGEMENT OF HEPATIC TUMORS. *(E.)* Shiu, M. H.; Fortner, J. G. (Mem. Sloan-Kettering Cancer Cent., New York, N. Y.). *Surg. Gynecol. Obstet.* 140(5):781-788, 1975.

4820 CLINICAL CHARACTERISTICS OF CHIEF HISTOLOGICAL FORMS OF LIVER CANCER. *(Rus.)* Fishzon-Ryss, Iu. I.; Mogilevskaia, I. A. (S. M. Kirov Acad. Mil. Med., Leningrad, USSR). *Vrach. Delo* (10): 31-34, 1974.

4821 ^{67}Ga-SCINTIGRAPHY AS AN AUXILIARY METHOD FOR DIFFERENTIATION OF MASS LIVER LESIONS. *(Ger.)* Blazek, G.; Mastnak, C.; Kahn, P.; Weiss, W.; Hanak, H. (Inst. Nucl. Med., Vienna, Austria). *Wien. Klin. Wochenschr.* 87(3):77-81, 1975.

4822 ULTRASONIC ANALYSIS OF LIVER CANCER. *(Fr.)* Melki, G. (Fac. Med., Rennes, France). *J. Radiol. Electrol.* 55(3):223-232, 1974.

4823 AN AGGREGATE-HEMAGGLUTINATION METHOD FOR DIAGNOSIS OF HEPATOCELLULAR CANCER AND TERATOBLASTOMAS. *(Rus.)* Biriulina, T. I.; Levina, D. M.; Elgort, D. A.; Gusev, A. I.; Iazova, A. K. (N. F. Gamaleia Inst. Epidemiol. Microbiol., Moscow, USSR). *Vopr. Onkol.* 20(11):1-120, 1974.

4824 CONTEMPORARY DIAGNOSIS OF CYSTS AND NEOPLASMS OF THE LIVER. *(Rus.)* Milonov, O. B.; Vasiliev, R. Kh.; Malyshenko, L. V. (All-Union Res. Inst. Clin. Exp. Surg., Moscow, USSR). *Khirurgiia (Mosk.)* (3):69-74, 1973.

4825 DIFFERENTIAL DIAGNOSIS OF BENIGN LIVER TUMORS (TWO CASES OF FOCAL NODULAR HYPERPLASIA). *(Ger.)* Mangold, G.; Kirschner, P.; Wagner, R.; Hohn, P. (Surg. Clin., Univ. Mainz, Germany). *Dtsch. Med. Wochenschr.* 100(6):241-243, 1975.

4826 DIAGNOSTIC PROBLEMS IN THE ETIOLOGY AND PATHOGENESIS OF PRIMARY HEPATOCELLULAR CANCER. *(It.)* Migneco, G.; Didato, A.; Tripi, S. (Inst. Gen. Clin. Med., Med. Ther., Univ. Palermo, Italy). *Boll. Ist. Sieroter. Milan* 53(1):79-87, 1974.

4827 RESECTION IN MULTIPLE HEPATIC LESIONS. *(Rus.)* Shapkin, V. S. (Vladivostok Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):77-80, 1974.

4828 RIGHT LOBECTOMY OF THE LIVER, WITH ISOLATION AND PERFUSION, IN LYMPHOSARCOMA. *(It.)* Mazzoni, G.; Di Martino, A.; Valli, C.; Demofonti, A.; Bancheri, C.; Gentili, B.; Pellegrini, S.; Melis, M. (Sch. Techn. Diagn. Histopathol., Univ. Rome, Italy). *Chir. Gastroenterol.* 7(2):211-219, 1973.

4829 A MULTIPLE CHEMOTHERAPEUTIC APPROACH TO THE MANAGEMENT OF HEPATOBLASTOMA. A PRELIMINARY REPORT. *(E.)* Holton, C. P.; Burrington, J. D.; Hatch, E. I. (Child. Hosp. Oncology Cent., Denver, Colo.). *Cancer* 35(4):1083-1087, 1975.

4830 ENZYME DETERMINATIONS IN THE DIAGNOSIS OF LIVER METASTASES. *(Sp.)* Garcia Bengoechea, M.; Ortega Vega, J.; Lanas Larranaga, E. (Nuestra Senora de Aranzazu Resid. Sanit., San Sebastian, Spain). *Rev. Clin. Esp.* 133(1):23-26, 1974.

4831 CONTROVERSIAL PROBLEMS IN HEPATECTOMY. *(Rus.)* Dederer, Iu. M.; Krylova, N. P.; Palamarchuk, V. P. (Altai Med. Inst., Barnaul, USSR). *Vestn. Khir.* 113(10):27-33, 1974.

4832 HEPATIC TRAUMA: FIVE CASES OF EMERGENCY MAJOR HEPATIC RESECTION. *(Fr.)* Atlas, H.; Allard, M.; Marcade, J. P. (Sacred Heart Hosp., Montpellier, France). *Rev. Fr. Gastroenterol.* (98):29-32, 35-38, 41-42, 1974.

4833 MAJOR HEPATECTOMY IN SURGICAL PRACTICE. *(Fr.)* Devic, G.; Garde, J.; Gelain, J.; Eymard, J. (no affil.). *Lyon Chir.* 71(1):3-8, 1975.

4834 MAJOR HEPATIC RESECTIONS IN CHILDREN. *(E.)* Martin, C. E.; Holcomb, G. W.; Foster, J. H.; (Vanderbilt Univ. Med. Cent., Nashville, Tenn.). *J. Pediatr. Surg.* 10(2):195-200, 1975.

4835 POSTOPERATIVE PROBLEMS AND MANAGEMENT AFTER HEPATIC RESECTION FOR BLUNT INJURY TO THE LIVER. *(E.)* Vajrabukka, T.; Bloom, A. L.; Sussman, M.; Wood, C. B.; Blumgart, L. H. (Glasgow R. Infirm., Scotland). *Br. J. Surg.* 62(3):189-200, 1975.

4836 BI-OCCLUSIVE CAVAL CATHETERIZATION IN MONITORING THE EFFICIENCY OF PORTOSYSTEMIC SHUNT OPERATIONS. *(It.)* Zannini, G.; Bracale, G. C.; Renda, A.; Masciariello, S. (2nd Fac. Med. Surg., Univ. Naples, Italy). *Chir. Gastroenterol.* 8(1):79-91, 1974.

4837 AN ENORMOUS INTRAHEPATIC SHUNT BETWEEN PORTAL VEIN AND HEPATIC ONE. *(E.)* Kozuka, S.; Sassa, R.; Kakumu, S. (Nagoya Univ. Sch. Med., Japan). *Angiology* 26(4):365-371, 1975.

4838 HEREDITARY ALPHA$_1$-ANTITRYPSIN DEFICIENCY ASSOCIATED WITH CONGENITAL EXTRAHEPATIC BILE DUCT HYPOPLASIA. *(E.)* Christen, H.; Bau, J.; Halsband, H. (Lubeck Med. Sch., Germany). *Klin. Wochenschr.* 53(2):90-91, 1975.

4839 STUDIES OF LIVER BIOPSIES IN THALASSEMIA MAJOR. *(Ger.)* Gmelin, K.; Rossner, J. A.; Feist, D. (Pathol. Inst., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Pathol.* 57:251-257, 1973.

4840 STUDY ON A CASE OF SICKLE CELL-THALASSEMIA. HEPATIC HISTOPATHOLOGY. *(It.)* Menardo, G.; Di Pietro, P.; Marenco, G.; Galli, E.; Canepa, M. (S. Corona Hosp. Inst., Pietra Ligure, Italy). *Pathologica* 66(955/956):187-194, 1974.

4841 HEPATIC INVOLVEMENT IN SCLERODERMA. *(Ger.)* Rau, R.; Pfenninger, K.; Lobsiger, M. (Rheumatol. Clin., Univ. Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 104(50):1877-1879, 1974.

4842 AGE-SPECIFIC FEATURES OF LIVER FUNCTION IN ESSENTIAL HYPERTENSION AND ATHEROSCLEROSIS. *(Rus.)* Fesenko, I. P. (Chernigov Reg. Hosp., USSR). *Vrach. Delo* (11):60-63, 1974.

4843 BENIGN FAMILIAL RECURRENT CHOLESTASIS (TYG-STRUP SYNDROME). *(Ger.)* Bosseckert, H.; Machnik, G.; Abendroth, K.; Eitner, K. (Med. Clin., Friedrich Schiller Univ., Jena, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(1):43-44, 1974.

4844 CHANGES IN THE FATTY ACID PATTERN OF TRIGLYCERIDES IN THE HEPATIC PARENCHYMA AND STORED LIPIDS IN DIABETICS AS A FUNCTION OF HEPATIC STEATOSIS. *(Ger.)* Singer, P.; Gnauck, G.; Thoelke, H.; Honigmann, G.; Buntrock, P.; Kettler, L. H. (Kaulsdorf Munic. Hosp., Berlin, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(1):27-35, 1974.

4845 TWO CASES OF ACUTE INTERMITTENT HEPATIC PORPHYRIA. *(Pol.)* Rogoz, J.; Kulakowski, S.; Lukomska, K. (Munic. Hosp., Sanok, Poland). *Wiad. Lek.* 27(24):2157-2160, 1974.

4846 CLINICAL AND HISTOLOGICAL FINDINGS IN 12 JAUNDICED PATIENTS WITH PULMONARY TUBERCULOSIS. *(It.)* Piccinino, F.; Manzillo, G.; Sagnelli, E.; Castaldo, M.; Balestrieri, G. G.; Arciello, G. (1st Fac. Med. Surg., Univ. Naples, Italy). *Fegato* 19(3):607-616, 1973.

4847 INTRAHEPATIC LITHIASIS. *(It.)* Benedetti-Valentini, S.; Benedetti-Valentini, G. (United Hosp., Rome, Italy). *Osp. Ital. Chir.* 27(1):17-23, 1974.

4848 CONTRIBUTION TO THE ETIOLOGY OF "REVERSIBLE HEPATIC DYSFUNCTION" (STAUFFER'S SYNDROME) ASSOCIATED WITH RENAL TUMORS. *(Ger.)* Girmann, G.; Gratzl, M.; Pees, H.; von Seebach, N. B.; Scheurlen, P. G. (Med. Clin. I, Univ. Saarland, Homburg/Saar, Germany). *Dtsch. Med. Wochenschr.* 100(10):480-484, 1975.

4849 HYPOGONADISM IN MALE PATIENTS WITH CHRONIC LIVER DISEASES. *(Ger.)* Szarvas, F.; Julesz, J.; Toth, I.; Faredin, I. (Med. Clin. I, Univ. Szeged, Hungary). *Z. Gesamte. Inn. Med.* 30(6):254-256, 1975.

4850 BIOPTICAL LIVER CHANGES IN FLORID HYPERTHYREOSIS. *(E.)* Lorenz, G.; Meng, W. (Pathol. Inst., Ernst Moritz Arndt Univ., Greifswald, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 22(1):22-25, 1975.

4851 THE EFFECT OF 14-DAY STARVATION ON MORPHOLOGICAL CHANGES IN THE LIVER IN PATIENTS WITH SIMPLE OBESITY. *(Pol.)* Tycowa, M.; Salwa-Kubasik, W. (Acad. Med., Poznan, Poland). *Pol. Arch. Med. Wewn.* 52(1):49-56, 1974.

4852 FREE AMINO ACIDS OF THE BLOOD SERUM AND CERTAIN PARAMETERS OF THE LIVER FUNCTION IN PATIENTS WITH CIRCULATORY DISORDERS. *(Rus.)* Sivkov, I. I.; Lebedeva, N. A. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Kardiologiia* 14(1):43-48, 1974.

4853 HEPATIC SECRETORY OBSTRUCTION WITH TOTAL PARENTERAL NUTRITION IN THE INFANT. *(E.)* Touloukian, R. J.; Seashore, J. H. (Yale Univ. Med. Sch., New Haven, Conn.). *J. Pediatr. Surg.* 10(3): 353-360, 1975.

See also, 4008, 4191, 4198, 4213, 4214, 4232, 4260, 4261, 4263, 4268, 4269, 4270, 4271, 4292, 4294, 4298, 4555, 4899, 4904, 4952, 5094, 5148, 5154.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

4854 STUDIES ON PHOTOTHERAPY IN NEWBORN INFANTS. INFLUENCE ON PROTEIN BINDING OF BILIRUBIN AND SALICYLATE AND ON ACTIVITY OF ACETYLSALICYLIC ACID ESTERASE. *(E.)* Windorfer, Jr. A.; Faxelius, G.; Boreus, L. O. (Karolinska Hosp., Stockholm, Sweden). *Acta Paediatr. Scand.* 64(2):293-298, 1975.

Protein binding both of bilirubin and salicylate before and after phototherapy was studied in newborns with hyperbilirubinemia. The effect of light on acetylsalicylic acid esterase was also studied, and for comparison, parallel experiments were done on light-treated rabbits and on light-treated blood and plasma *in vitro*. Newborns (15) were subjected to phototherapy for 2-4 days for hyperbilirubinemia; five neonates from the same ward were used as control patients. Blood analyses were made before and after phototherapy. An increase was observed in hematocrit values and in the activity of the erythrocyte enzyme acetylsalicylic acid esterase. The elevation of the enzyme activity also could be produced in light-treated rabbits and *in vitro* after illumination of blood from adult volunteers. The binding of bilirubin to serum albumin and of salicylate to plasma proteins was not altered, nor were the concentrations of albumin or total proteins in plasma. The results indicate that light does not increase the unbound fraction of bilirubin in blood.

4855 THE PLACE OF NON-CONJUGATED BILIRUBIN AND SERUM ALBUMIN DOSAGES IN THE SURVEILLANCE OF PREMATURE ICTERUS. *(Fr.)* Satge, P.; Colin, J.; Voyer, M.; Crumiere, C.; Charlas, J. (Inst. Puericult., Paris, France). *Arch. Fr. Pediatr.* 31 (2/3):201-213, 1974.

4856 THE ELEMENTS OF THERAPY IN THE CRIGLER-NAJJAR SYNDROME. *(It.)* Bartolozzi, G.; Ciampolini, M.; Gentilini, P. (Inst. Clin. Pediatr., Univ. Florence, Italy). *Fegato* 19(2):457-468, 1973.

See also, 4116, 4117, 4122, 4123.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

4857 HALOTHANE-RELATED HEPATITIS. A CLINICAL STUDY OF TWENTY-SIX CASES. *(E.)* Moult, P. J. A.; Sherlock, S. (R. Free Hosp., London, England). *Q. J. Med.* 44(173):99-114, 1975.

The clinical details of halothane-induced hepatitis were reviewed in 26 cases. The mean body weight was 68.4 kg for the women and 74.4 kg for the men. No patients were considered where hepatitis might have been attributable to another anesthetic. None had either alcoholism or chronic liver disease. With the exception of four laparotomies and four craniotomies, the operations were relatively minor. All 26 patients became jaundiced within 12 days of halothane anesthesia. One patient gave a history of jaundice after an anesthetic 8 yr previously. Excepting two patients, all had 2, 3, or 4 exposures to halothane within a 3-month period. Eight patients had blood transfusions, seven of them within the previous month. Eight were given phenothiazine drugs and two coricosteriods postoperatively, and 16 were given antibiotics and two diphenylhydantoin preoperatively. Eight patients had delayed and unexplainable pyrexia as previously described in halothane-induced hepatitis. Treatment of fulminant hepatic failure was by dextrose infusion, oral neomycin, enemas, and vitamin K. All 11 patients who died were in fulminant hepatic failure. Two were treated by exchange transfusion. Jaundice in the 15 survivors lasted from 1-12 weeks. None developed any clinical or biochemical evidence of liver disease. The minimum

thrombo-test in the fatal cases was 15% or less, while only three of the survivors had less than 15%. Mean body weight was 79.8 kg in fatal cases as opposed to 64.9 kg for survivors. All fatal cases became jaundiced within 7 days of the last anesthetic, while all who became jaundiced after this time survived. It is concluded that halothane-induced hepatitis should be regarded as a clinical entity, and prognosis is related to obesity and rapidity with which jaundice appears postoperatively.

4858 HEPATIC COMPLICATIONS OF ANTITUBERCULOUS THERAPY. *(E.)* Rossouw, J. E.; Saunders, S. J. (Groote Schuur Hosp., Capetown, South Africa). *Q. J. Med.* 44(173):1-16, 1975.

The frequency of hepatic reactions to drug treatments for pulmonary tuberculosis and patterns for each drug (sensitization time, associated hypersensitivity, biochemical findings and prognosis) were investigated. Records of 7492 patients admitted to two hospitals for pulmonary tuberculosis between 1960 and 1973 were included in the study. Drug hepatitis was diagnosed if the patient became clinically jaundiced or had at least two abnormal liver function tests. Patients with abnormal baseline liver function tests, or who had a history of alcoholism, recent blood transfusion, liver disease, contact with infectious hepatitis or exposure to other drugs were excluded. The reaction was assigned to a particular drug: if the patient was on the drug for at least 3 weeks preceeding the reaction; if after withdrawal, rechallenge reproduced the reaction; if after withdrawal, rechallenge with other drugs did not reproduce the reaction; or after withdrawal of the suspected drug but with continuation of other drugs, the reaction settled. Of the 7492 cases, there were 24 cases of hepatitis (0.32%); 14 cases from another hospital were added. Para-aminosalicyclic acid was implicated in 16 cases, while in 12 cases it was either this agent or isonicotinic hydrazide. The latter alone was responsible for three cases, ethionamide for one, and multiple drugs for three. The three isonicotinic hydrazide patients were over 50 yr of age, as compared to five in this age bracket in the remaining 35 cases. The time between the starting of the drug and the first symptom was under 90 days in all cases. Most (89%) of the patients had premonitory symptoms preceeding the jaundice, including rash (23 patients), pyrexia, and gastrointestinal disturbances. Clinical jaundice developed in all but five. Skin lesions were found in 80%. Pyrexia was common in the para-aminosalicyclic acid (69%) and para-aminosalicyclic acid plus isonicotinic hydrazide (85%) groups, and lymphadenopathy was found in 46% of both. Hepatomegaly was almost universal. No patients with para-aminosalicyclic acid hepatitis died, while there was a 25-33% mortality in the other groups. It is suggested that such patients be kept under close surveillance and drug therapy stopped as soon as warning symptoms appear.

4859 THE COMPETING RISKS OF TUBERCULOSIS AND HEPATITIS FOR ADULT TUBERCULIN REACTORS. *(E.)* Comstock, G. W.; Edwards, P. Q. (Sch. Hyg. Public Health, Johns Hopkins Univ., Baltimore, Md.). *Am. Rev. Respir. Dis.* 111(5):573-577, 1975.

The competing risks of tuberculosis and isoniazid-associated hepatitis were estimated according to the best evidence now available. Tuberculosis risks were derived from two sets of data on adult reactors to 5 TU of purified protein derivative: average annual tuberculosis case rates during a 20-yr period among reactors in Muscogee County, Georgia, and Russell County, Alabama; and average annual case rates during a 10-yr period among reactors given placebo in mental hospitals in Georgia, Massachusetts, and Wisconsin. Hepatitis risks were derived from U.S. Public Health Service information on persons over 20 yr who had taken isoniazid for up to 1 yr. Lifetime tuberculosis risks/1000 reactors and the number of tuberculosis cases preventable by isoniazid prophylaxis are shown according to race, sex, and age (25-65 yr). Estimated hepatitis rates/1000 persons are given for specified ages, as are the expected number of cases among 1000 reactors completing 1 yr of isoniazid treatment. The ratio of tuberculosis cases preventable by treatment to hepatitis cases occurring during treatment is specified according to race, sex, and age. The probability of developing hepatitis during 1 yr of treatment with isoniazid ranges from 2.4/1000 adults younger than 35 yr to 19.2/1000 at 55-64 yr. It is concluded that the upper age limit for isoniazid prophylaxis should be set at 45 yr for reactors without additional risk factors. With additional risk factors, such as recent conversion or household exposure, abnormal chest roentgenogram, diabetes, or long-term corticosteroid therapy, it is probably wise to give preventive treatment to all but the very old.

4860 DIAGNOSIS OF ACUTE SYPHILITIC HEPATITIS. *(Rus.)* Zeldin, V. E.; Storozhakova, I. A.; Vishneevtskii, A. M.; Kelner, R. I. (Cent. Res. Inst. Dermatol. Syphilol., Moscow, USSR). *Klin. Med. (Mosk.)* 52(11):133-135, 1974.

A case is reported of acute syphilitic hepatitis in a 43-yr-old man hospitalized with complaints of anemia, loss of appetite, nausea, jaundice, itching and subfebrile fever. No pathology of the urinary system was observed. The liver was enlarged 4 cm beyond the costal arch. Blood values upon admission were Hgb 13 g%; erythrocytes 4,100,000 mm^3; color index 0.92; leukocytes 8,000; ESR 22 mm/hr. Urine values were: nitrogen 22 mg%; bilirubin 4.72 mg%; cholesterol 252 mg%; SGOT 48 U; SGPT 50 U; whole protein 8.17 g%; albumin 39.6%; globulin α_1 = 9.4%; α_2 = 13.6%; β = 15.3%; γ 22.1%. The albumin-globulin coefficient was 0.66. The case was diagnosed as chronic hepatitis and prednisolone treatment (15 mg/day) was prescribed with glucose, cocarboxylase and vitamins. On the 16th day of hospitalization moist papules were observed in the anal region. Wassermann reaction was ++++ (titre 1:320). Kahn's and Sachs-Witebsky tests were strongly positive. Roseola elements were observed on the body skin; papule-like formations, on the palms and feet. The diagnosis was recurrent secondary syphilis and acute syphilitic epithelial hepatitis. Treatment by penicillin (14,000,000 U) was prescribed.

The blood and urine values returned to normal 1 month after the treatment began. Wassermann and precipitation tests continued to be strongly positive. The patient was discharged in a satisfactory condition. It is indicated that clinically and morphologically, acute syphilitic hepatitis has much in common with viral hepatitis except for certain specific features: 1) jaundice is accompanied by eruptions on the skin and mucous membranes; 2) the process in the liver is quickly reversed as a result of antisyphilitic treatment; and 3) a sharp drop in the albumin content and an increase in the globulin content is observed.

4861 HEPATITIS DUE TO AFLATOXICOSIS: AN OUTBREAK IN WESTERN INDIA. *(E.)* Krishnamachari, K. A. V. R.; Nagarajan, V.; Bhat, R. V.; Tilak, T. B. G. (Indian Counc. Med. Res., Hyderabad). *Lancet* 1(7915):1061-1063, 1975.

An epidemic of hepatitis in parts of western India was reported; the syndrome was characterized by icterus, rapid onset of signs of portal hypertension, ascites, and a high mortality rate. The disease affected man and dogs and was associated with the consumption of maize heavily contaminated with *Aspergillus flavus*; 106 of 397 patients died. In five maize samples obtained from affected households, the aflatoxin levels ranged between 6.25 and 15.6 ppm; thus, the patients would have ingested 2-6 mg aflatoxin/day for several weeks. Detectable levels of aflatoxin B_1 were found in 2 of 7 serum samples. A specimen of liver obtained at necropsy showed bile duct proliferation and giant cells. The observations indicate that staple foodgrains can become sufficiently infected to result in disease, and that there is a need for divising means of averting such contamination.

4862 CHOLESTATIC HEPATITIS AFTER ADMINISTRATION OF FURAN DERIVATIVES. *(E.)* Engel, J. J.; Vogt, T. R.; Wilson, D. E. (Abraham Lincoln Sch. Med., Univ. Ill., Chicago). *Arch. Intern. Med.* 135 (5):733-735, 1975.

A case was reported of a patient who developed jaundice following nitrofurantoin therapy and subsequently developed a second episode of liver injury following intravaginal administration of a mixture of nifuroxine and furazolidone. Liver function tests following nitrofurantoin therapy for a urinary tract infection (300 mg/day, 7 days) showed liver enzyme values of: SGOT, 121 U; SGPT, 256 U; alkaline phosphatase, 350 IU. The patient was treated for vaginal trichomoniasis 1 week later and received two vaginal suppositories, each containing furazolidone (5 mg) and nifuroxime (7.5 mg); liver enzyme values that day were SGOT, 49 U; SGPT, 96 U; and alkaline phosphatase, 169 IU. The enzyme levels 4 days later rose to: SGOT, 256 U; SGPT, 325 U; and alkaline phosphatase, 760 IU. Fractionation of the alkaline phosphatase showed that it was of liver origin. After discontinuing the suppository therapy, the liver enzymes slowly returned to normal. The data suggest cross-sensitivity between the two furan derivatives and that such cross-sensitivity of chemically related compounds be considered even when they are administered by different routes.

4863 THREE CASES OF SEVERE ACUTE HEPATIC FAILURE OCCURRED AFTER REPEATED HALOTHANE ANESTHESIA. *(E.)* Kosaka, Y.; Matsuda, Y.; Tagawa, S.; Terada, M.; Akeda, S.; Nakagiri, S.; Tameda, Y.; Tsujita, E.; Kondo, I.; Shiomi, Y.; Kawarada, R.; Okuda, Y. (Mie Sch. Med., Tsu, Japan). *Mie. Med. J.* 24(1):81-92, 1974.

4864 DRUG INDUCED SUBACUTE HEPATITIS TREATED WITH EXCHANGE BLOOD TRANSFUSION. *(E.)* Kosaka, Y.; Matsuda, Y.; Tagawa, S.; Terada, M.; Nakagiri, S.; Kondo, I.; Tameda, Y.; Shiomi, Y.; Tsujita, E.; Kawarada, R.; Okuda, Y.; Kawakita, K.; Akeda, S. (Mie Univ. Sch. Med., Tsu, Japan). *Mie Med. J.* 24(1):93-100, 1974.

4865 HEPATOTOXICITY OF RIFAMPICIN AND ISONIAZID IN CHILDREN. *(E.)* Casteels-Van Daele, M.; Igodt-Ameye, L.; Corbeel, L.; Eeckels, R. (Dep. Pediatr., Univ. Louvain, Belgium). *J. Pediatr.* 86 (5):739-741, 1975.

4866 CADMIUM - ITS IN VIVO DETECTION IN MAN. *(E.)* McLellan, J. S.; Thomas, B. J.; Fremlin, J. H.; Harvey, T. C. (Dep. Phys., Univ. Birmingham, England). *Phys. Med. Biol.* 20(1):88-95, 1975.

4867 LIVER FUNCTION DISORDERS INDUCED BY ANTIBACTERIALS USED IN THE TREATMENT OF CHRONIC PULMONARY TUBERCULOSIS. *(It.)* Gammarrota, V.; Grassi, P. E. (Spec. Prov. Hosp. Tuberc., Ente, Italy). *Fegato* 19(3):617-626, 1973.

4868 THE INCIDENCE OF THE LIVER DAMAGE IN PREGNANT SICILIAN WOMEN. *(It.)* Carollo, F.; Barba, G.; Cadilli, G. (Inst. Clin. Obstet. Gynecol., Univ. Palermo, Italy). *Rev. Ital. Gynecol.* 55(4): 341-350, 1974.

4869 NON-PARASITIC LIVER CYSTS: REPORT OF A CASE. *(Fr.)* Bekaert, S.; Afschrift, M.; Verdonk, G.; Kunnen, M. (Acad. Hosp., Univ. Gand, Belgium). *Ann. Radiol. (Paris)* 17(5):445-449, 1974.

See also, 4846, 5137, 5138, 5139, 5142, 5144, 5145, 5146, 5149.

4870 THERAPEUTIC EFFECT OF GLUCOCORTICOIDS ON THE COURSE OF VIRAL HEPATITIS IN CHILDREN WITH REGARD TO THE PRESENCE OF AUSTRALIA ANTIGEN. *(Pol.)* Kossakowski, R.; Sokolewicz, E.; Szelag, O. (Acad. Med., Bialystok, Poland). *Pediatr. Pol.* 48 (10):1197-1202, 1973.

Fifteen children (aged 3 months to 12 years) with viral hepatitis and serum Australia antigen (AuAg) and 15 children of the same age with viral hepatitis but without AuAg were treated with 1 mg/kg/day prednisone for 3-6 weeks. AuAg-positive children had a longer history of the disease and were ill longer (up to 147 days) before therapy than AuAg-negative children (maximum duration 71 days). However, the thymol turbidity test, SGPT and SGOT activity, and hepatomegaly were less pronounced in AuAg-positive than in AuAg-negative children. After prednisone therapy the Au Ag-negative children showed rapid clinical and biochemical improvement, while AuAg-positive children showed only transient improvement; 2 weeks after discontinuation of the therapy the serum transaminase activities and hepatomegaly recurred.

4871 CELL-MEDIATED IMMUNITY IN ACUTE AND CHRON- HEPATITIS. *(E.)* Wands, J. R.; Perrotto, J. L.; Alpert, E.; Isselbacher, K. J. (Harvard Med. Sch., Boston, Mass.). *J. Clin. Invest.* 55(5):921-929, 1975.

Peripheral lymphocytes from 22 patients (18 men and 4 women, aged 18-65 yr) with acute hepatitis, chronic active hepatitis, or chronic persistent hepatitis and from nine controls were tested for *in vitro* cytotoxicity and blast transformation. Of the 22 patients, 17 were positive and five were negative for hepatitis-B surface antigen (HB_sAg). Cytotoxicity was measured by ^{51}Cr release into the medium from labeled Chang cells after 6-hr incubation with peripheral lymphocytes at a lymphocyte target cell ratio of 200:1. Concomitant 72-hr incubation studies were performed toassess thymus cell-dependent (T) lymphocytic function as measured by concanavalin A (Con A)-stimulated incorporation of tritiated thymidine (blast transformation) and by cytotoxicity. Lymphocytes from patients with acute hepatitis were significantly cytotoxic to Chang cells compared with control lymphocytes (12.71% *versus* 2.71%). Lymphocytes from patients with acute and chronic hepatitis were less cytotoxic when incubated with autologous and homologous HB_sAg-positive serum; and lymphocytes from patients with HB_sAg-positive and negative acute, chronic active hepatitis, or chronic persistent hepatitis were as cytotoxic as normal controls when stimulated with Con A. Lymphocytes from chronic active hepatitis patients on prednisone therapy (30 or 50 mg/day for 2.5 months) showed marked depression of cytotoxicity when stimulated with Con A. These studies show that patients with acute hepatitis have circulating T lymphocytes that are capable of causing destruction of Chang liver cells; that patients with acute and chronic hepatitis have a serum factor(s) that inhibits spontaneous and induced lymphocyte cytotoxicity and blast transformation; and that prednisone treatment appatently inhibits lymphocyte toxicity in patients with chronic active hepatitis.

4872 PROPHYLAXIS OF POSTTRANSFUSION HEPATITIS: BIOCHEMICAL TESTS FOR RESEARCH IN BLOOD DONORS. *(Fr.)* Plomteux, G. (Lab., Med. Chem., Liege, Belgium). *Ann. Gastroenterol. Hepatol. (Paris)* 10(5):401-411, 1974.

Patients with acute viral hepatitis (110) were checked weekly until 34 parameters relating to hepatic function indicated normalization of hypertransaminasemia. The frequency of perturbation of the parameter and degree of global sensitivity of the test in the population studied were calculated for each parameter. It was found that the highest frequencies of perturbations were for SGPT (100.00%), SGOT (98.21%), and ornithine carbamoyltransferase (OCT), (85.71%). The coefficients of sensitivity for the tests for the three substances were, resp., 118.43, 79.29, and 50.24. In 2 yr, 5,397 U of blood were transfused and assayed for degree of perturbation in the concentration of SGPT, SGOT, and OCT. One or more of the enzymes occurred in abnormal concentrations in 6.32% of the donors, but only 2.68% showed perturbation of the concentrations of all three enzymes and in the relative order determined in the tests on hepatitis patients. The latter units of blood were classified as biochemically suspect if the value of the SGPT was at least 100 mIU. A number of patients who had received both normal and suspect blood were followed up. A statistically significant difference was found in the subsequent evolution of plasma concentrations of the three enzymes in the recipients of normal *versus* suspect blood. In 2 examples of the development of recipients of suspect blood, the plasma concentrations of the three enzymes increased tremendously by the fourth or fifth week after transfusion, and then gradually returned to normal.

4873 HOST COMPONENTS IN HEPATITIS B ANTIGEN. *(E.)* Burrell, C. R. (Edinburgh Univ. Med. Sch., Scotland). *J. Gen. Virol.* 27(2):117-126, 1975.

Radioimmunoassays were performed to determine if 20-25 nm hepatitis antigen (HB Ag) particles are intrinsically composed of normal serum components, modified by hepatitis B virus infection in the host to express a unique virus-specific antigenic character, or whether these particles represent primarily virus-code material, associated in some preparations with contaminating serum components. Purified ^{125}I-labeled 20-25 nm HB Ag particles gave low affinity immunoprecipitation reactions with antisera to several normal serum components; the reactions were immunologically distinct from the reaction due to the classical HB Ag surface determinant. These additional antigenic determinants were acid-stable and tightly bound to the particles; they could not be released by treatment with Tween 80 or ether, but were removed by protease digestion with preservation of particle integrity. It was not possible to distinguish wheth-

er they were due to the presence of trace amounts of partly denatured serum components, or to a weak cross-reaction with antigens present in normal serum. Some cross-reaction with antigens in normal serum, in particular involving the superficial carbohydrate moieties of HB Ag, would not be surprising in sensitive assays. No evidence was found for native antigenic material, present in normal serum or normal liver cells, being integral to the structure of these particles.

4874 SERUM IMMUNOGLOBULIN M (IgM) VALUES IN VIRAL HEPATITIS PATIENTS. *(E.)* Ene, M.; Roman, D.; Luca, V.; Duda, R. (Iasi Inst. Med. Pharm., Rumania). *Rev. Med. Chir. Soc. Med. Nat. Iasi* 78(3): 659-662, 1974.

Serum immunoglobulin M (IgM) levels were measured by radial immunodiffusion in 80 patients with jaundice, of whom 68 had viral hepatitis and 12 (controls) had other hepatobiliary diseases. In 12 viral hepatitis patients with Australia antigen (HB_sAg) and eight without HB_sAg but with a long period of incubation (more than 60 days), the mean serum IgM value was 197 mg/100 ml or close to the control value of 144 mg/100 ml. These 20 patients were assumed to have virus B hepatitis. The remaining 48 viral hepatitis patients without HB_sAg had a significantly higher IgM level of 273 mg/100 ml. This group was considered to have virus A hepatitis. The results suggest that determination of serum IgM within the first 10 days of jaundice would permic differentiation of the two forms of viral hepatitis according to immunologic criteria.

4875 PROGNOSTIC SIGNIFICANCE OF SUBACUTE HEPATIC NECROSIS IN ACUTE HEPATITIS. *(E.)* Ware, A. J.; Eigenbrodt, E. H.; Combes, B. (Univ. Texas Southwest. Med. Sch., Dallas). *Gastroenterology* 68 (3):519-524, 1975.

A retrospective analysis was made of 57 patients treated during the period 1967-1973 with the lesion of subacute hepatic necrosis in the setting of acute hepatitis. Of the 57 patients, 40% were male and 60% female, with 63% under 30 yr of age, and 21% over 40 (age range of 14-88 yr). Epidemiology included contact with hepatitis (25%), blood transfusion (4%), i. v. drug use (26%), and hepatotoxic drugs (12%). Of the 49 patients so tested, 67% carried Australia antigen (HB_sAg) in their sera. Excluding the seven patients considered to have a drug-related liver injury, the incidence of HB_sAg positivity rises to almost 80%. The drugs implicated as the probable etiologic agent in these seven were halothane (three), aldomet (two), isoniazid, and propylthiouracil. Of the 57 patients, 14 were lost to follow-up but were doing very well when last seen. One paitent died with massive hepatic necrosis 6 weeks after her biopsy had shown the lesion of subacute hepatic necrosis. The remaining 42 were observed from 2-73 months (average 19 months). Eight patients progressed to chronic active liver disease, one of whom died in hepatic failure after 14 months of continuous disease. In the other 34 patients, liver function tests returned to normal and remained so without therapy. In managing patients with subacute hepatic necrosis, high dose steroid therapy was instituted on making the diagnosis unless clinical and biochemical improvement was already underway. Thus, steroids were more likely given to the most seriously ill patients. The patient who progressed to massive necrosis did so despite high dose prednisolone therapy. Not one of the eight patients who developed chronic active liver disease, however, received steroid therapy during their acute illness, although a number of them have since been so treated. Thus the results of this study do not support the view that steroid therapy in acute hepatitis may actually encourage the development of chronic disease. Repeat biopsies from 10 months to 6 yr after the initial biopsy in nine of the 34 patients who underwent biochemical resolution showed two of them with inactive postnecrotic cirrhosis. The incidence of these complications developing in patients with subacute hepatic necrosis was about 30% in this study. These findings add qualitative support to the position that liver biopsy findings bear important prognostic value in patients with acute hepatitis.

4876 AUSTRALIA-ANTIGEN (HB_sAg) AND HEPATITIS IN CHILDHOOD. *(Ger.)* Hellwege, H. H. (Univ. Clin. Pediatr., Hamburg-Eppendorf, Germany). *Monatsschr. Kinderheilkd.* 122(12):893-899, 1974.

Transient Australia antigen (HB_sAg) was detected in sera from two children of 12 with acute sporadic hepatitis who had not been exposed to potential parenteral infective agents. One of these had a long lasting close contact with a chronic HB_sAg carrier. Five of seven children with acute hepatitis following blood transfusions were HB_sAg-positive. In all cases of acute hepatitis, whether HB_sAg positive or negative, the illness was transient, and persistence of HB_sAg never occurred. Five of eight children with chronic active hepatitis were HB_sAg carriers. Parenteral exposure to potentially virus B-containing infective agents could be excluded in every case. Antinuclear antibodies were detected in the sera of two HB_sAg-negative patients. During 15 months sera of 71 polytransfused children with hemophilia were tested serially. In this period five children developed acute hepatitis: two of them were chronic HB_sAg carriers; and in one boy HB_sAg could be detected only for a short time. When serum bilirubin and transiminase levels normalized, one chronic HB_sAg carrier became negative. Two patients with hepatitis were known as chronic HB antibody (HB_sAb) carriers. Antibodies against HB_sAg were detected in sera of 26 patients with hemophilia without liver disease. More boys than girls suffering from acute and chronic hepatitis could be observed. The boys were predominating in both HB_sAg-positive and negative groups. The incidence of HB_sAg detection in these children suggests that Virus B-infection may play an important role in the pathogenesis of chronic active hepatitis in childhood.

4877 A STATISTICAL CONTRIBUTION ON HEPATITIS AND THE AUSTRALIA ANTIGEN AMONG YOUNG 'ITINERANTS' TO QUEBEC. *(Fr.)* Maurice, P.; Valet, J. P.; Rousseau, C.; Matte, J. (Laval Univ. Hosp. Cent., Quebec, Canada). *Union Med. Can.* 104(3):436-439, 1975.

4878 FREQUENCY OF THE AUSTRALIA ANTIGEN (HB_S AG) AMONG THE POPULATION OF THE CITY OF QUEBEC AND EAST OF QUEBEC PROVINCE TABULATED BY RADIOIMMUNOLOGICAL TECHNIQUES. *(Fr.)* Valet, J. P.; Rousseau, C.; Ackerman, H. W.; Matte, J. (Laval Univ. Hosp. Cent., Quebec, Canada). *Union Med. Can.* 104(3):439-443, 1975.

4879 RELATION BETWEEN HEPATITIS B ANTIGEN AND SOME CLINICAL HISTOLOGICAL AND LABORATORY DATA IN CHRONIC AGGRESSIVE HEPATITIS. *(It.)* Guisti, G.; Piccinino, F.; Galanti, B.; Ruggiero, G.; Manzillo, G.; Sagnelli, E.; Utili, R. (Fac. Med. I, Univ. Naples, Italy). *Rass. Clin. Sci.* 50(2):48-53, 1974.

4880 INVESTIGATIONS CONCERNING THE FIXATION OF HEPATITIS B-ANTIGEN (HBAg) ON PERIPHERAL LYMPHOCYTES AND ISOLATED HEPATOCYTES IN PATIENTS WITH INFLAMMATORY LIVER DISEASES. *(Ger.)* Arnold, W.; Meyer zum Buschenfelde, K. H.; Hopf, U.; Kordbarlag, C. (Med. Clin. II, Johannes Gutenberg Univ., Mainz, Germany). *Klin. Wochenschr.* 53(5):231-235, 1975.

4881 ENCAPSULATING PERI-HEPATITIS. REPORT OF FOUR CASES. *(Fr.)* Debray, C.; Leymarios, J.; Cerf, M.; Husson, J. M. (Bichat Hosp., Paris, France). *Sem. Hop. Paris* 51(5):289-297, 1975.

4882 COMPARITIVE EPIDEMIOLOGICAL AND CLINICAL STUDIES ON HEPATITIS A AND B IN THE MAGDEBURG AREA. *(Ger.)* Herms, G.; Kramer, G. (Reg. Hosp., Magdeburg-Altstadt, Germany). *Z. Gesamte. Inn. Med.* 30(5):183-187, 1975.

4883 DETERMINATION OF ENDOGENOUS VITAMIN B_{12} BY THE RADIOISOTOPE METHOD IN CHILDREN WITH VIRUS HEPATITIS. *(Pol.)* Kaczmarski, M. (Acad. Med., Bialystok, Poland). *Pediatr. Pol.* 50(1):37-45, 1975.

4884 HEMOSTASIS SYSTEM IN VIRUS HEPATITIS. PART II. FIBRINOLYTIC SYSTEM. *(Pol.)* Rosiek, B. (Acad. Med., Lodz, Poland). *Pol. Arch. Med. Wewn.* 52(1):41-47, 1974.

4885 HEMOSTATIC SYSTEM IN VIRUS HEPATITIS. PART III. SOLUBLE FIBRIN MONOMER COMPLEXES AND FIBRINOGEN DEGRADATION PRODUCTS IN PATIENTS WITH SEVERE VIRUS HEPATITIS. *(Pol.)* Rosiek, B. (Acad. Med., Lodz, Poland). *Po. Arch. Med. Wewn.* 52(2):173-178, 1974.

4886 HEMOSTASIS SYSTEM IN VIRUS HEPATITIS. *(Pol.)* Rosiek, B. (Acad. Med., Lodz, Poland). *Pol. Arch. Med. Wewn.* 52(2):179-186, 1974.

4887 HEMOSTASIS SYSTEM IN VIRUS HEPATITIS. PART I. BLOOD CLOTTING SYSTEM. *(Pol.)* Rosiek, B. (Acad. Med., Lodz, Poland). *Pol. Arch. Med. Wewn.* 52(1):31-40, 1974.

4888 EFFECT OF INFECTIOUS HEPATITIS ON THE COURSE OF PRIMARY HYPERLIPOPROTEINAEMIA. *(Cz.)* Kucerova, I.; Brodanova, M. (Dep. Gen. Med., Charles Univ., Prague, Czechoslovakia). *Cs. Gastroenterol. Vyz.* 28(6):397-401, 1974.

4889 FUNCTIONAL AND HISTOLOGICAL CONDITION OF GASTRIC MUCOSA IN PATIENTS AFTER INFECTIOUS HEPATITIS. *(Rus.)* Barba, E. I.; Vasilyuk, V. N.; Boiko, M. S. (Ternopol Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11):97-101, 1974.

4890 PATHOGENETIC THERAPY IN CHILDREN WITH VIRAL HEPATITIS. *(Rus.)* Nisevich, N. I.; Uchaikin, V. F.; Novokshonov, A. A.; Yakob, L. E. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 18(12):3-9, 1973.

4891 THE CONTENT OF HISTAMINE IN THE BLOOD AND HISTAMINE-BINDING CAPACITY OF PLASMA PROTEINS IN VIRAL HEPATITIS. *(Rus.)* Sorinson, S. N.; Mikhailenko, A. A. (Kishinev Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11):89-93, 1974.

4892 THE ULTRASTRUCTURE OF GIANT CELLS IN HEPATITIS: ETIOPATHOGENETIC CONSIDERATIONS. *(Sp.)* Varela Nunez, R.; Diaz-Flores, L.; Ortiz Urdian, G.; Abad, J.; Sanchez Salgado, G. (Sch. Pathol. Histol. Anat., Santiago, Chile). *Rev. Clin. Esp.* 133 (1):11-16, 1974.

4893 STATISTICAL STUDY ON THE AUSTRALIA ANTIGEN IN THE REGION WEST OF QUEBEC. *(Fr.)* Guevin, R. M.; Brown, G.; Taliano, V. (Red Cross Transfus. Cent., Montreal, Canada). *Rev. Fr. Gastroenterol.* (97):53-58, 1974.

4894 EVOLUTION OF ISOENZYMES OF LACTATE DEHYDROGENASE DURING VIRAL HEPATITIS (A STUDY OF 130 CASES). *(Fr.)* Laverdant, C.; Bon, R.; Cristau, P.; Essioux, H.; Molinie, C.; Kassem, A.; Gendron, Y.; Jezequel, Y. (Serv. Gastroenterol., HIA Begin, Saint-Mande, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(5):389-399, 1974.

4895 EXTRACORPOREAL PERFUSION OF PIG LIVER IN THE TREATMENT OF HEPATIC COMA. *(It.)* Zucchetti, F.; Butti, A.; Fianchini, A.; Pigliucci, G. M.; Bianco, A. (Clin. Gen. Med. Med. Ther., S. Coure Cathol. Univ., Rome, Italy). *Chir. Patol. Sper.* 21(1):63-79, 1973.

4896 PRELIMINARY RESULTS ON THE RECOVERY OF THE AUSTRALIA ANTIGEN FROM FECES AND URINE OF PATIENTS WITH VIRAL HEPATITIS AND OTHER LIVER DISEASES. *(It.)* Zacchi, T. (United Hosp., Trieste, Italy). *Boll. Soc. Ital. Cardiol.* 53(1):52-58, 1974.

4897 HISTOLOGICAL ASPECTS OF THE INTESTINAL MUCOSA IN ACUTE AND CHRONIC HEPATITIS. *(It.)* Pastore, G.; Buongiorno, R.; Dentico, P.; Fera, G.; Schiraldi, O. (Inst. Inject. Dis., Univ. Bari, Italy). *Boll. Ist. Sieroter. Milan.* 53(2):402-414, 1974.

4898 HISTOLOGICAL CHANGES OF HEPATOCYTES AS A POSSIBLE SIGN FOR CARRIER OF HB-ANTIGEN. *(Ger.)* Korb, G.; Huppertz, A. (Weiden Munic. Hosp., Upper Palatinate, Germany). *Klin. Wochenschr.* 52 (23):1132-1133, 1974.

See also, 4285, 4793, 4846, 4925.

LIVER AND BILIARY TRACT

Chronic Hepatitis

4899 CLOTTING AND FIBRINOLYTIC ACTIVITY OF BLOOD IN CHRONIC LIVER DISEASE. *(Pol.)* Kaniak, J.; Kotschy, M.; Kowal-Gierczak, B.; Plamieniak, L. (Acad. Med., Wroclaw, Poland). *Pol. Tyg. Lek.* 29(38): 1621-1624, 1974.

The blood clotting system and fibrinolysis were investigated in 60 patients with chronic liver disease. These patients consisted of 30 with liver necrosis, 15 with chronic hepatitis, and 15 with liver metastases of malignant neoplasms. Patients with cirrhosis showed reduced blood clotting and increased fibrinolytic activity (decrease in fibrinogen, prothrombin, factors VII and IX, a prolonged Stypvencephalin time, low plasminogen values, shortened fibrinolysis time, and reduced values for antiplasmin and antithrombin). Patients with liver metastases showed increased blood clotting and decreased fibrinolytic activity, as indicated by increased serum thromboplastic activity in the Biggs-Douglas test: increased values of prothrombin and fibrinogen; somewhat low values for plasminogen; and a prolonged fibrinolysis time. Results in patients with chronic hepatitis were not specific and were in the lower normal range. Low values for prothrombin, factors VII and IX, and fibrinogen could be a manifestation of decreased synthesis by injured liver cells. Low values of fibrinogen, plasminogen, antiplasmin, and antithrombin could also result from intensified activation of fibrinolysis often observed in chronic liver disease.

4900 SYSTEMIC MANIFESTATIONS ASSOCIATED WITH CHRONIC AGGRESSIVE HEPATITIS. *(Fr.)* Geubal, A. (Saint-Pierre Univ. Clin. Louvain, Belgium). *Acta Gastroentero. Belg.* 37(4-5):192-206, 1974.

A retrospective study was made of 53 patients with histologically confirmed chronic aggressive hepatitis from clinical, serological, and pathological information available when the hepatitis was diagnosed or later. Systemic manifestations occurred more than twice as often in women (21 of 28) as in men (9 of 25). The most common systemic manifestations involved the skin (19 cases) and joints (15), but they were also found in the endocrine system (9 cases), kidneys (4), blood (3), gastrointestinal tract (2), lymphatic system (2), muscles (1), heart (1), and pleura and pericardium (1); 3 patients had paraproteinemia. Associated disorders consisted of clinical or preclinical diabetes (18 cases), neuropsychiatric disorders (8), peptic ulcer (5), and allergies (8). The severity of systemic manifestations was not related to the extent of liver malfunction nor to immunoglobulin levels. Antithyroid antibodies were detected in 62% of the patients, antigastric antibodies in 28%, and Australia antigen in 9 of the 30 studied. The severity of systemic manifestations was greatest in patients who were negative for Australia antigen on serum immunoelectro-osmophoresis. It was confirmed that anti-smooth muscle antibodies and Australia antigen are not found in the same patient, and antinuclear factor was associated with Australia antigen in only 1 case. Total hemolytic complement and B_1A globulin levels were significantly decreased in patients with systemic manifestations. No correlation was found between B_1A globulin concentrations and the Australia antigen titer. Complement titers were affected both by the extent of liver malfunction and by the existence of systemic lesions, particularly renal ones. In most patients, complement titers decreased during exacerbations of chronic aggressive hepatitis. The extrahepatic lesions found in these patients and the associated serological anomalies suggest that chronic aggressive hepatitis is an autoimmune disease.

4901 CELLULAR AND HUMORAL IMMUNITY TO HEPATITIS-B SURFACE ANTIGEN IN ACTIVE CHRONIC HEPATITIS. *(E.)* Lee, W. M.; Reed, W. D.; Mitchell, C. G.; Galbraith, R. M.; Eddleston, A. L. W. F.; Zuckerman, A. J.; Williams, R. (King's Coll. Hosp. Med. Sch., London, England). *Br. Med. J.* 1(5960):705-708, 1975.

Although hepatitis-B surface antigen (HB_sAg) may be persistently present in the serum in a few cases of active chronic hepatitis, the cause of the disease in most patients in unknown. Studies carried out on 39 HB_sAg-negative patients with active chronic hepatitis revealed the presence of cell-mediated immunity to HB_sAg in 24 (62%), suggesting a high frequency of previous infection with hepatitis-B virus. Hepatitis-B surface antibody was detectable by ra-

dioimmunoassay in six patients, in all of whom HB_sAg complexes were present in the serum on electron microscopy. Patients with HB_sAg-positive active chronic hepatitis (12) were also studied and eight, including all those untreated at the time, showed a cellular response to the antigen. Evidence of sensitization to a liver-specific cell surface lipoprotein was found with similar frequency in both groups. These results are consistent with the hypothesis that hepatitis-B virus is important in initiating the disease in many cases of active chronic hepatitis and that sensitization to the liver cell membrane antigen is the autoimmune process responsible for perpetuation of the liver injury.

4902 HAEMOLYTIC COMPLEMENT, ANTICOMPLEMENTARY ACTIVITY AND HEPATITIS B ANTIGEN IN SERA OF PATIENTS WITH CIRRHOSIS OF THE LIVER. *(E.)* Kelkar, S. S.; Karhade, N. V.; Kotwal, S. E. (Med. Coll., Aurangabad, India). *Indian J. Med. Res.* 63(1):41-44, 1975.

Sera from 50 patients with liver cirrhosis (34 men and 16 women, mean age 34.5 yr) were studied for the presence of hepatitis B antigen (HB Ag), anticomplementary activity, and level of hemolytic complement. The latter two tests were done within 2 hr of sample collection to reduce artifacts. Of the 50 sera, 15 samples (30%) showed HB Ag by the immunoelectroosmophoretic technique but none contained hepatitis B antibody (HB Ab). Only two patients showed anticomplementary activity; both were negative for HB Ag and HB Ab. In the remaining 48 cases, hemolytic complement levels (mean 3.6 U) were significantly lower than levels (mean 7.2 U) in sera from 25 healthy controls. Further analysis showed that the hemolytic complement levels were significantly reduced in both HB Ag-positive and negative cases compared with normal controls. Differences in complement levels between the HB Ag-positive and negative groups were not significant. Reduction of hemolytic complement may be due to reduced synthesis by the damaged liver or to its utilization in possibly pathogenic mechanisms. It is postulated that the first view is correct and that a cell-mediated response is involved in hepatic damage in HB Ag-associated cirrhosis.

4903 SERUM IMMUNOGLOBULINS AND ANTIBODIES IN RELATIVES OF PATIENTS WITH CHRONIC ACTIVE HEPATITIS (CAH). *(E.)* Chiaramonte, M.; Borrelli, A.; Sturniolo, G. C.; Fagiolo, U.; Naccarato, R. (Inst. Med. Pathol., Univ. Padua, Italy). *Rend. Gastroenterol.* 6(3):205-206, 1974.

In an attempt to determine if a familiar abnormality of the immune response is involved in the development of chronic active hepatitis from acute hepatitis, 78 close relatives of nine patients with chronic hepatitis (five HB Ag-positive and four HB Ag negative) were investigated for HB Ag (counterelectrophoresis), serum immunoglobulins (radial immunodiffusion), antinuclear and anticytoplasma antibodies (RA-latex test), OCT, serum albumin and γ-globulin. All relatives were HB-negative. Serum antibodies were not significantly increased compared with controls. Immunological changes were found only in families of two HB Ag-positive and two HB Ag-negative patients. Mild biochemical changes were also evident in some subjects.

4904 ASYMPTOMATIC AUTOIMMUNE LIVER DISEASE IN SCLERODERMA. *(E.)* Gupta, M. M.; Warnes, T. W.; Watson, L. (Univ. Coll. Hosp., London, England). *Postgrad. Med. J.* 51(593):185-189, 1975.

A case of scleroderma with asymptomatic autoimmune liver disease is reported. The 54-yr-old female patient presented with features of CRST syndrome (calcinosis, Raynaud's phenomenon, sclerodactyly, and telangiectasia). Routine investigation revealed elevated plasma alkaline phosphatase (63 King-Armstrong U/100 ml). Separation of the isoenzymes of alkaline phosphatase by electrophoresis on polyacrylamide gels showed that the elevated enzyme was of hepatic origin; the presence of antimitochondrial, antinuclear, and smooth muscle antibodies in the serum suggested an associated autoimmune liver disease, which was confirmed by liver biopsy. Biopsy showed slight cholestasis and inflammatory cell infiltration around the portal system with bile ductule proliferation and reticulin increase extending into lobules. The histologic appearance was compatible with active chronic hepatitis progressing to cirrhosis. Routine measurement of serum alkaline phosphatase in all patients with scleroderma is indicated as a simple screening procedure for autoimmune liver disease.

4905 IMMUNOSUPPRESSIVE THERAPY IN CHRONIC HEPATITIS IN CHILDREN. *(E.)* Rabenda, C.; Rudkowski, Z. (Med. Acad., Wroclaw, Poland). *Mater. Med. Pol.* 6(3):202-206, 1974.

After failure of corticotherapy, six children (aged 2-9 yr) with chronic hepatitis were treated with immunosuppressive drugs (6-mercaptopurine, cyclophosphamide, and azathioprine). Permanent remissions lasting from 2 months to 4 yr were obtained in five cases. No side effects of the drugs were observed apart from moderate leukopenia and thrombocytopenia. Infections were not more frequent or more severe in the treated children. Varicella in one child ran a mild course. Case reports are presented for three children treated with 6-mercaptopurine followed by cyclophosphamide (0.5-1.0 mg/kg/day) or cyclophosphamide plus aziothioprine (0.8 mg/kg/day). 6-Mercaptopurine was without effect in one child who subsequently responded to cyclophosphamide therapy. Liver and spleen decreased in size as early as 2 weeks after immunosuppressive therapy, followed by disappearance of antinuclear antibodies from the serum and reversion of biochemical indexes to normal. Further trials of immunosuppressive therapy in children with chronic hepatitis appear warrented.

4906 IMMUNOSUPPRESSIVE TREATMENT OF CHRONIC HEPATITIS. *(Pol.)* Stempien, R.; Bergiel, A.; Fabianowski, J. (Acad. Med., Lodz, Poland). *Pol. Tyg. Lek.* 29(37):1583-1586, 1974.

A total of 29 patients (12 women and 17 men, aged 30-50 yr; av. 43.6 yr) with confirmed chronic hepatitis were treated with azathioprine (100-150 mg/day for 2-4 weeks followed by a maintenance dose of 50 mg/day) and prednisolone (15-20 mg/day). The duration of therapy ranged from 6-14 months; most patients are still being treated. The progress of disease was arrested in 23 of the 29 patients, and in some cases a complete remission was obtained. Marked clinical and biochemical improvement occurred in 6, some improvement in 17, no change in 5, and deterioration in 1. A decrease in hepatomegaly was observed in 20 of 29; of splenomegaly in 12 of 14; of ascites in 4 of 4; disappearance of telangiectasia in 5 of 10; and of palmar erythema in 5 of 13. Bilirubin levels returned to normal in 11 of 16 and improved in 2; albumin levels in 6 of 14 and 2 of 14, resp.; protein levels in 4 of 7 and 1 of 7, resp.; gamma globulin in 6 of 29 and 10 of 19, resp.; the thymol turbidity test in 7 of 29 and 13 of 29, resp.; and SGPT in 18 of 25 and 4 of 25, resp. Viral hepatitis preceded the disease in 24 cases; in 19 cases it occurred 4-18 months before immunosuppressive therapy was instituted. There were no serious side effects of treatment: 6 patients had brief episodes of upper respiratory infection and 10 showed transient decreases in the WBC or platelet count.

4907 ALPHA-MERCAPTOPROPIONYLGLYCINE (THIOLA) IN THE TREATMENT OF CHRONIC LIVER DISEASES. *(It.)* Nuti, M. (Italian Cent. Gastroenterol., Univ. Rome). *Policlinico. Sez. Med.* 81(1):55-62, 1974.

4908 COMPARATIVE ELECTRON MICROSCOPE INVESTIGATIONS IN CHRONIC PERSISTENT AND CHRONIC AGGRESSIVE HEPATITIS. *(Pol.)* Biczysko, W.; Meissner, R.; Zengteler, G.; Juszczyk, J. (Acad. Med., Poznan, Poland). *Pol. Arch. Med. Wewn.* 52(3):229-238, 1974.

4909 SURGICAL THERAPY OF CHRONIC HEPATITIS. *(Rus.)* Milonov, O. B.; Movchun, A. A. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (9):67-71, 1974.

4910 BIO- AND IMMUNOLOGICAL STUDIES IN A CASE OF CHRONIC ACTIVE HEPATITIS IN A 12 YEAR OLD CHILD. *(Fr.)* Storcz, J.; Baumgartner, I.; Fulop, T.; Otvos, I.; Par, A.; Kaiser, E. (Pediatr. Clin., Pecs, Hungary). *Pediatrics* 10(46):49-58, 1974.

4911 CLINICAL SIGNIFICANCE OF MULTIPLE PROJECTION HEPATOSCANNING IN CHRONIC HEPATITIS. *(Rus.)* Sukirno, M. S.; Suchkov, A. V.; Iudin, L. A. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Klin. Med.* 52(8):107-113, 1974.

See also, 4265, 4266, 4793, 4801, 4802, 4871, 4897, 4923, 5099.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

4912 HOST FACTORS CAUSING INCREASED SUSCEPTIBILITY TO INFECTION IN PATIENTS WITH LAENNEC'S CIRRHOSIS. *(E.)* Andersen, B. R. (Abraham Lincoln Sch. Med., Univ. Illinois, Chicago). *Ann. NY Acad. Sci.* 252:348-352, 1975.

Infection is one of the common precipitating factors of hepatic coma in patients with cirrhosis and is the immediate cause of death of about 1/2 of patients with Laennec's cirrhosis. Facets of the host-defense mechanisms that appear to be most important in preventing the pyogenic infections that predominate in cirrhotics are the reticuloendothelial system, antibodies, complement, and neutrophilic granulocytes. Current knowledge about the functional capacity of these systems in cirrhotics is reviewed with emphasis on the complement system. Granulocytes of cirrhotic patients apparently have no significant functional defect and there is clearly no deficiency in antibodies function. Studies of reticuloendothelial function in cirrhotic rats demonstrated that i.v. injected bacteria were normally cleared from blood, although there was a delay in bacterial killing in the animals. A study of 12 cirrhotic patients at a VA hospital showed a decrease in complement components, especially C3 (7/12), C4 (1/2), and C3 proactivator (4/12). Of 11 patients tested for total hemolytic complement, eight had below-normal levels. These complement deficiencies may provide a less than optimal chemotactic and phagocytic stimulus for the granulocytes of the cirrhotic patient. The poor chemotactic response of the patients to their own serum was initially attributed to the depression of complement components. However, normal serum added to patient serum did not correct the problem. A serum inhibitor of chemotaxis was demonstrated in most patients with advanced Laennec's cirrhosis; the inhibitor was quite labile and quickly lost at room temperature.

4913 FOLATE DEFICIENCY IN THE ALCOHOLIC - ITS RELATIONSHIP TO CLINICAL AND HAEMATOLOGICAL ABNORMALITIES, LIVER DISEASE AND FOLATE STORES. *(E.)* Wu, A.; Chanarin, I.; Slavin, G.; Levi, A. J. (Northwick Park Hosp., Harrow, England). *Br. J. Haematol.* 29(3):469-478, 1975.

Clinical and laboratory observations were made on 84 patients (56 men and 28 women, aged 27-78 yr) regularly taking more than 80 g of alcohol/day. Macrocytosis was present in 84.5%, but only 13% were anemic. Of 57 bone marrow samples, 27 were abnormal, and 20 showed megaloblastic changes, most-

ly mild in degree. Serum, RBC, and liver folate levels were reduced in 28%, 35%, and 31% of the patients, resp. Liver folate deficiency was more severe in those patients, more often female, who had inadequate diets or drank wine and spirits rather than beer. The amount of alcohol consumption and extent of liver damage did not affect folate status. These findings suggest that folate deficiency is common among alcoholics in England. However, this study emphasizes that alcohol can cause macrocytic and megaloblastic changes by two separate mechanisms, one secondary to folate deficiency, another by a direct toxic effect. The direct toxic effect plays a more significant role in alcoholics who take an adequate diet, as did most of the patients in this study. Many of the patients had macrocytosis, sometimes with megaloblastosis, in the absence of folate deficiency, emphasizing the direct toxic effect of alcohol on the developing erythroblast. The findings of this study are not compatible with the suggestion that negative folate balance always precedes megaloblastic conversion.

4914 THE IgA/TRANSFERRIN RATIO IN CIRRHOSIS: DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE. *(Fr.)* Fruchart, J. C.; Jaillard, J.; Sezille, G.; Paris, J. C.; Rault, C.; DeBuire, B. (Munic. Hosp., Lille, France). *Nouv. Presse. Med.* 3(28):1751-1752, 1974.

4915 CHYLOMICRONEMIA INDUCED BY ALCOHOL IN TYPE IV HYPERLIPOPROTEINEMIA. *(Fr.)* Brun, D.; Moorjani, S.; Lupien, P. J.; Belanger, G. (Laval Univ. Hosp. Cent., Quebec, Canada). *Rev. Fr. Gastroenterol.* (97):21-23, 1974.

4916 CHOLESTASIS IN ALCOHOLIC HEPATITIS. *(Ger.)* Brunner, H. (Med. Clin. II, Univ. Vienna, Austria). *Leber Magen Darm* 4(1):32-36, 1974.

4917 AUTOIMMUNE PHENOMENA IN ALCOHOLIC CIRRHOSIS. *(E.)* Zinneman, H. H. (Minneapolis VA Hosp., Minn.). *Am. J. Dig. Dis.* 20(4):337-345, 1975.

See also, 4780, 4787.

LIVER AND BILIARY TRACT

Cirrhosis

4918 INTRAVENOUS VASOPRESSIN IN PATIENTS WITH PORTAL HYPERTENSION: ADVANTAGES OF CONTINOUS INFUSION. *(E.)* Thomford. N. R.; Sirinek, K. R. (Ohio State Univ. Coll. Med., Columbus). *J. Surg. Res.* 18(2):113-117, 1975.

Continuous infusion and bolus injection of vasopressin were compared in reduction of portal vein pressure and adverse hemodynamic side effects. The study included seven patients with Laennec's cirrhosis who had hemorrhages from esophageal varices. Polyethylene catheters were inserted into the portal vein, superior vena cava, and one radial artery to monitor pressures and determine cardiac output. After 72 hr each patient was daily stabilized for 30-60 min and tested for 60 min for the hemodynamic effects of the vasopressin. Recordings were made at 0, 5, 15, 30, 45, and 60 min. Bolus administration consisted of 15 U lysine-vasopressin in 100 ml 5% dextrose given in 5 min. Infusion consisted of 0.4 U lysine-vasopressin/ml 5% dextrose at 100 ml/hr. The two types of administration were alternated daily. The bolus injections caused severe cramping and abdominal pain within 60 sec, and prompted defecation and urination within 5 min, as well as causing nausea in all patients. Premature artrial contractions occurred in seven patients and persisted as long as 30 min. During the injection, systolic pressure increased briefly from 117 to 138 mm Hg. A significant brachycardia occurred and persisted for 60 min. Average portal pressure decreased from 366 mm saline to 294 mm saline after 5 min and gradually increased to 315, 329, and 341 mm after 15, 30 and 60 min, resp. Average cardiac output decreased from 6.7 to 5.6 liters/min at 30 min and was still significantly decreased after 60 min. Continuous infusion caused minimal to moderate abdominal cramping within 2 min and prompted defecation and urination within 5 min. Three became nauseated and three had occasional premature artrial contractions. Systolic pressure increased transiently during the first 5 min from 121-142 mm Hg. Brachycardia occurred within 5 min and persisted for 60 min. Average portal pressure decreased from 365 to 280, 269, and 261 mm saline at 5, 30, and 60 min, resp. Average cardiac output decreased from 6.1 liters/min to 4.8 liters/min at 5 min, but after this, values rose to a less than significant output reduction. Portal vein reduction pressures at 15, 30, 45, and 60 min were significantly lower after infusion than after bolus injection. It is concluded that infusion causes significantly greater reduction of portal pressure, less discomfort, and less reduction in cardiac output than bolus injection.

4919 THE EFFECT OF DIURETIC TREATMENT ON RED CELL AND PLASMA SODIUM CONCENTRATION IN PATIENTS WITH HEPATIC CIRRHOSIS AND ASCITES. *(E.)* Afifi, F. (INSERM, Beaujon Hosp., Clichy, France). *Biomedicine* 23(2):68-72, 1975.

The sodium content of RBC and plasma in 10 patients with hepatic cirrhosis and ascites was measured be-

fore, during, and after diuretic treatment. The results were compared with findings in 37 normal subjects and 42 untreated cirrhotic patients with ascites. Before diuretic treatment the mean RBC sodium content in the 10 patients with ascites was 9.95 mEq/kg; expressed as mEq/kg dry weight and mEq/kg RBC water, the sodium content was 27.15 and 15.02, resp. During diuretic treatment, the mean RBC sodium content decreased significantly to 6.27 mEq/kg, 18.85 mEq/kg dry weight, and 10.15 mEq/kg RBC water. The mean pretreatment plasma sodium concentration was 135.95 mEq/kg, which significantly decreased during treatment to 129 mEq/kg. After cessation of diuretic treatment and the disappearance of ascites, RBC and plasma sodium levels returned to normal. *In vitro* ^{22}Na exchange between plasma and RBC of cirrhotic patients with ascites remained unmodified during diuretic treatment. These results indicate that RBC in patients with cirrhosis complicated by ascites are a parameter of intracellular sodium retention. Their increased sodium content is diminished by diuretic treatment.

4920 ADDITIONAL DATA ON HEPATIC FUNCTION TESTS IN CYSTIC FIBROSIS. *(E.)* Feigelson, J.; Pecau, Y.; Cathelineau, L.; Navarro, J. (Trousseau Hosp., Paris, France). *Acta Paediatr. Scand.* 64(2): 337-344, 1975.

Over a 3-yr period, various liver function tests were performed on 50 patients (20 men and 30 women, aged 1-26 yr) with cystic fibrosis, of whom nine had multilobular cirrhosis. Tests indicating cytolysis, cholestasis, and cellular insufficiency were carried out together with measurements of serum prothrombin and immunoglobulins. Test results were generally abnormal in the patients with cirrhosis. Two distinct biochemical types of cirrhosis were distinguished, one clearly cholestatic and the other a more cellular type. The first type was characterized by elevated γ-glutamyl transpeptidase levels, significant cytolysis with raised ornithine transcarbamylase and B_{12} levels, normal IgA, and normal or high transferrin levels giving an IgA:transferrin ratio of less than 1.0. The second type of cirrhosis was characterized by consistently high IgA levels with normal or low transferrin levels giving an IgA:transferrin ratio greater than 1.9. Of the noncirrhotic patients, 50% showed a temporary increase of cytolysis (elevated ornithine transcarbamylase and B_{12} levels) and cholestasis (elevated leucine aminopeptidase). Three of the 50 patients, including one with cirrhosis, developed viral hepatitis. In those without cirrhosis, all tests returned to normal and remained so for more than 2 yr. It is concluded that hepatic disfunction remains merely latent in cystic fibrosis.

4921 AN ATYPICAL CYTOPLASMIC ANTIBODY IN CHRONIC LIVER DISEASES. DETECTION BY INDIRECT IMMUNE FLUORESCENCE. *(Ger.)* Storch, W. (Med. Clin., Karl-Marx Univ. Leipzig, Germany). *Z. Inn. Med.* 29 (22):940-942, 1974.

Sera from 400 patients with chronic liver diseases (cirrhosis, chronic aggressive hepatitis), and from 400 normal subjects were tested for an atypical cytoplasmic antibody by indirect immune fluorescence, using rat kidney slices as an antigenic substrate for incubation. Atypical cytoplasmic antibody was found in 12 patients with chronic liver diseases, in one normal subject, and titers over 1:400 were found in four patients without liver disease. No sex-specific differences in the occurrence were established. The occurrence of the antibody was highest in patients with liver cirrhosis. Australia antigen was usually absent. There was no correlation between the occurrence of the atypical cytoplasmic antibody and the seriousness of the disease. Fluorescence exclusively of the proximal tubular epithelium was found. The typical pattern of the fluorescence clearly differed from those observed in the presence of other antibodies. The antibody is not specific of the species, and belongs to the IgG fraction. A single absorption on liver mitochondria and microsomes did not suppress the fluorescence.

4922 HEPATIC ARTERIES AND ARTERIAL CIRCULATION IN LIVER CIRRHOSIS. *(E.)* Takahashi, T.; Tezuka, F. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 113(2):113-128, 1974.

Hepatic arterial circulation in normal and cirrhotic livers was analyzed by morphometrical treatment of the arterial system. Autopsy materials obtained from 32 cases of liver cirrhosis, three cases of Banti's syndrome, and 98 control cases were used. The cirrhotic group included postnecrotic and portal annular cirrhosis; biliary cirrhosis and cirrhosis with primary hepatic carcinoma were excluded. Arterial blood flow of the liver was estimated from the radius R_t of the proper hepatic arteries. The arterial flow Q was calculated from the equation $Q = q \cdot R_t^n$, where q and n are the organ-independent constants of the arterial system. The mean arterial flow Q in cirrhotic livers was significantly larger than in normal livers, and there was a striking individual difference in Q in cirrhotic livers. Arterial flow Q was closely correlated with the volume of interstitial tissue, but not with the volume of parenchymal tissue, in cirrhotic livers. In Banti's syndrome, the estimated hepatic arterial flow was within the normal range. It is concluded that the increased supply of arterial blood in liver cirrhosis was due solely to abundant intrahepatic shunts. Compensation of decreased portal flow by increased arterial supply was improbable.

4923 PLASMA AND RED BLOOD CELL PHOSPHOLIPIDS IN CHRONIC LIVER DISEASES. *(E.)* Cantoni, L.; Curri, S. B.; Andreuzzi, P.; Rocchetti, P. (Hosp. Maggiore Ca' Granda, Milan, Italy). *Clin. Chim. Acta* 60(3):405-408, 1975.

Total phospholipid levels and levels of phosphatidylcholine, lysophosphatidylcholine, phosphatidylethanolamine and phosphatidylserine, and sphingomyelin were determined in the plasma and RBC of 58 patients with chronic liver disease and 12 controls by quantitative extraction and thin-layer chromatography. The liver disease in 12 patients with cirrhosis was compensated, in 41 decompensated, the remainder having

chronic aggressive hepatitis. Total phospholipid levels were significantly reduced in both the plasma and RBC of the patients with chronic liver disease, as compared with controls. Plasma sphingomyelein was significantly decreased in liver disease, while RBC showed a marked increase in lysophosphatidylcholine and a significant decrease in phosphatidylethanolamine and phosphatidylserine. It is suggested that in chronic liver disease, RBC lipid disturbances follow more closely those of the hepatocyte than those in plasma.

4924 ANTIBODY TITRE CHANGES AND SKIN REACTIVITY IN PATIENTS WITH LIVER CIRRHOSIS UNDERGOING PORTOCAVAL SHUNT OPERATION. *(E.)* Johansen, K. S.; Johansen, T. S.; Leerhoy, J. (State Serum Inst., Copenhagen, Denmark). *Acta Med. Scand.* 197(3):225-228, 1975.

Antibody titers to common antigens, and cell mediated immunity, were studied in cirrhotic patients undergoing portocaval shunts. Sera were collected from 10 patients with alcoholic and post-necrotic liver cirrhosis, and examined for HI antibodies to rubella and vaccinia, and CF antibody to cytomegalovirus. Normal blood donors (21) served as controls. Antistreptolysin O, antistreptococcal hyaluronidase, diptheria toxoid, *Candida* precipitin, syphilis reactions, *Salmonella typhi* O and H and *S. paratyphi* O and H were also determined. Skin tests were performed with *Candida albicans* extract, diptheria toxoid B antigen, streptokinase and tuberculin. Eight patients undergoing cholecystectomy served as controls for the skin tests, and for antibody titers to diptheria toxoid and *Candida albicans precipitins*. The rubella (7/10) and vaccinia (8/10) HI antibodies, diptheria (6/10) antitoxin, and antistreptolysin O (3/10) titers were significantly higher in cirrhotics than in controls (5/21, 2/13, 0/8, and 0/21, resp.). Total number of elevated antibody titers was 36 of 140 as opposed to 9 of 194 in the controls. About one month after the portocaval shunt, eight of the 10 patients had a positive titer index and none was negative. In one patient after surgery, rubella and vaccinia titers jumped from 20 to 160 and from 32 to 256, resp. The skin test reactions to the four antigens used were lower in the cirrhotics than in the controls, though significantly only with the diphtheria toxoid and streptokinase. After portocaval shunt, mean antibody titers increased with a concomitant decrease in skin test reactions, while there were no titer changes and normal indurational increases after cholecystectomy. It is concluded that elevated antibody titers to many common bacterial, fungal, and viral antigens are found in cirrhotic patients.

4925 THE ROLE OF IMMUNOLOGIC FACTORS IN THE PATHOGENESIS OF CHRONIC ACTIVE HEPATITIS. *(Pol.)* Bergiel, A. (Med. Acad., Lodz, Poland). *Przegl. Epidemiol.* 28(2):131-137, 1974.

Immunologic factors in progressive active hepatitis, primary biliary cirrhosis and cryptogenic cirrhosis are reviewed. Progressive hepatic diseases are frequently considered to be autoimmune diseases because of the absence of definite cause, more frequent occurrence among young women, chronic course with exacerbations, familial occurrence associated with other autoallergic diseases, hypergammaglobulinemia, high level of immunoglobulins, presence of autoantibodies in serum, presence of LE factor, lymphocytic infiltrations and positive response to treatment with immunosuppressive and glycocorticoid drugs. The role of T and B lymphocytes in the production of autoantibodies is discussed and stressed that both humoral and cell mediated immunity have to be considered in chronic liver diesease. Antibodies to smooth muscle have been detected in 39-50% of patients with progressive active hepatitis and in 50% of primary biliary cirrhosis; antimitochondrial antibodies in 25-30% and 83-93% resp; antinuclear antibodies in 15-26% and 25%, resp. The role of Australia antigen (HB_sAg) or its complexes in the pathogenesis of progressive hepatitis has not been elucidated but HB_sAg-positive cases frequently have a different course and seem to have a better prognosis. "Mediolan" antigen, a lipoprotein, has been detected in serum when liver damage is present. It has been detected in 38% of patients with biliary cirrhosis, 26% of aggressive hepatitis and 30% of cryptogenic cirrhosis. Both HB_sAg and "Mediolan" antigen show a negative correlation to other autoimmune factors such as antinuclear and anti-smooth muscle antibody levels and show no correlation to antimitochondrial antibodies. The role of humoral immunity and toxicity of circulating autoantibodies has not been established and their detection has only diagnostic significance in chronic liver diseases. Cell mediated immunity, on the other hand, seems to be important in the pathogenesis of these diseases. It is hypothesized that the T lymphocytes of subjects with normal cellular immunity can become sensitized to HB_sAg antigen and attack liver cells containing HB_sAg. Subjects with decreased cellular immunity become HB_sAg carriers and show only slight liver damage.

4926 ANEMIA AND HEPATIC CIRRHOSIS. *(Sp.)* Musso, A. M.; Kremenchuzky, S.; Rochna Viola, E. M.; Burucua, J. E. (Jose de San Martin Hosp. Sch., Univ. Buenos Aires, Argentina). *Medicina (B. Aires)* 34(2):89-100, 1974.

4927 DIGESTIVE HEMORRHAGE IN THE CIRRHOTIC. *(Sp.)* Munoz Monteavaro, C.; Mescia, C. (Pereira Rossell Hosp., Montivideo, Uruguay). *Cir. Urug.* 44(4):278-288, 1974.

4928 PATHOGENESIS OF ASCITES FORMATION IN LIVER CIRRHOSIS. *(Ger.)* Arbeiter, G.; Meyer-Burg, J. (Med. Clin. Polyclin., Free Univ. Berlin, Germany). *Leber Magen Darm* 4(6):299-307, 1974.

4929 POSTERIOR MEDIASTINAL VENOUS MASSES IN PATIENTS WITH PORTAL HYPERTENSION. *(E.)* Moult, P. J. A.; Waite, D. W.; Dick, R. (R. Free Hosp., London, England). *Gut* 16(1):57-61, 1975.

4930 THE USEFULNESS OF THE GALATIN AND XYLOSE TOLERANCE TESTS IN INVESTIGATION OF DIGESTION AND ABSORPTION IN PATIENTS WITH CONGESTIVE LIVER CIRRHOSIS. *(Pol.)* Zydowicz, L.; Adamiak, T.; Chojnacki, J.; Kowalczyk, L. (Mil. Acad. Med., Lodz, Poland). *Pol. Arch. Med. Wewn.* 51(6):612-618, 1974.

4931 MEDICAL TREATMENT OF ASCITES. *(Fl.)* De Groote, J.; Deruyttere, M. (St. Raphael Acad. Hosp., Louvain, Belgium). *Tijdschr. Gastroenterol.* 17(2):94-102, 1974.

4932 LYMPHOVENOUS ANASTOMOSIS IN THE NECK OF PATIENTS WITH LIVER ASCITIC CIRRHOSIS. *(Rus.)* Shereni, P.; Madyar, Z.; Sabo, D. (Centr. Inst. Traumatol., Budapest, Hungary). *Khirurgiia (Mosk.)* (3):100-102, 1974.

4933 ADDITIONAL ARTERIALIZATION OF THE LIVER IN PORTACAVAL SHUNT. *(Rus.)* Burger, K.; Zimmerman, X. B.; Hubner, R.; Hagemann, E.; Munchov, R.; Tausch, V. (Sharite Med. Centr., Humboldt Univ., Berlin, Germany). *Khirurgiia (Mosk.)* (3):74-76, 1974.

4934 α_1-ANTITRYPSIN-DEFICIENCY: COMBINATION OF PULMONARY EMPHYSEMA AND LIVER CIRRHOSIS IN INFANCY. *(Ger.)* Kaiser, D.; Rennert, O. M.; Joller-Jemelka, H.; Gotze, H.; Sollberger, H.; Kehrli, P. (Pediatr. Clin., Univ. Bern, Switzerland). *Klin. Wochenschr.* 53(3):117-124, 1975.

4935 THE USE OF LACTULOSE IN THE TREATMENT OF CHRONIC PORTO-SYSTEMIC ENCEPHALOPATHY. *(Por.)* Sette, Jr., H.; Conte, V. P.; Bettarello, A. (Fac. Med., Univ. Sao Paulo, Brazil). *Arq. Gastroenterol.* 11(4):217-222, 1974.

4936 PRELIMINARY OBSERVATIONS OF HEMODYNAMICS IN CIRRHOTICS TREATED WITH L-DOPA. *(It.)* Cagossi, M.; Bianco, S.; Cagossi, M.; Butti, A.; Lojacono, L. (Inst. Gen. Clin. Surg. Surg. Ther., Sacra Cuore Cathol. Univ. Rome, Italy). *Chir. Patol. Sper.* 21(2):148-161, 1973.

4937 HEPATIC VENO-OCCLUSIVE DISEASE. TWO CASES IN QUEBEC. *(Fr.)* Auger, C.; Lefebvre, R. (Hotel Dieu, Montreal, Canada). *Rev. Fr. Gastroenterol.* (97):43-52, 1974.

4938 EMERGENCY TREATMENT FOR MASSIVE HEMORRHAGE FROM RUPTURED ESOPHAGEAL VARICES IN THE CIRRHOTIC PATIENT. *(Fr.)* Van Cangh, P. J.; Otte, J. B.; Mahieu, P.; Moriau, M.; Fiasse, R.; Kestens, P. J. (St. Pierre Clin., Cathol. Univ. Louvain, Belgium). *Acta Gastroenterol. Belg.* 37(6):259-272, 1974.

4939 END-TO-END CAVOSPLENIC ANASTOMOSIS FOR THE TREATMENT OF PORTAL HYPERTENSION IN CHILDREN. *(Ger.)* Burger, K.; Zimmermann, H. B.; Otto, H. H. (Charite Hosp., Humboldt Univ., Germany). *Zentralbl. Chir.* 99(41):1292-1295, 1974.

See also, 4264, 4266, 4308, 4429, 4618, 4741, 4793, 4801, 4802, 4807, 4814, 4837, 4838, 4881, 4899, 4902, 4914, 5090, 5099, 5184.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

4940 A TEST FOR PATENCY OF THE CYSTIC DUCT IN ACUTE CHOLECYSTITIS. *(E.)* Eikman, E. A.; Cameron, J. L.; Colman, M.; Natarajan, T. K.; Dugal, P.; Wagner, Jr., H. N. (Johns Hopkins Med. Inst., Baltimore, Md.). *Ann. Intern. Med.* 82(3):318-322, 1975.

A radioactive tracer procedure was used to evaluate the patency of the cystic duct in 39 patients with acute abdominal pain. The gallbladder is first stimulated to empty by i.v. injection of chloecystokinin (75 Ivy dog U). After 30 min, either ^{125}I-rose bengal (150 μCi) or ^{99m}Tc dihydrothioctic acid (2 mCi) are injected and accumulation of radioactivity in the liver and gallbladder regions is monitored by external gamma emission imaging. Diagnostically significant images were recorded between 60-90 min after injection of tracer. Ten patients who subsequently proved to have acute cholecystitis failed to accumulate gallbladder radioactivity. The remaining 29 patients with a variety of other diseases all showed visualization of the gallbladder. There were no significant side effects or complications of the procedure, and it is concluded that this technique may be a useful adjunct to the clinical and roentgenographic evaluation of patients with acute abdominal pain.

4941 BILIARY LIPID OUTPUT DURING THREE MEALS AND AN OVERNIGHT FAST. I. RELATIONSHIP TO BILE ACID POOL SIZE AND CHOLESTEROL SATURATION OF BILE IN GALLSTONE AND CONTROL SUBJECTS. *(E.)* Northfield, T. C.; Hofmann, A. F. (Mayo Clin., Rochester, Minn.). *Gut* 16(1):1-17, 1975.

Using a duodenal perfusion technique, the biliary output of bile acids, phospholipid, and cholesterol was measured hourly during 3 meals and an overnight fast in 7 white patients (3 men and 4 women, aged 42-69 yr)

with radiolucent gallstones in a functioning gallbladder, and in 7 healthy controls without gallstones, closely matched for age, sex, and weight. Before the perfusion, bile acid kinetics were defined by an isotope dilution procedure, and the biliary lipid composition of fasting gallbladder bile was determined. Total daily biliary lipid output was similar in gallstone and control subjects, and was unrelated to cholesterol saturation of fasting gallbladder bile and to bile acid pool size. There was an inverse relation between the size and recycling frequency of the bile acid pool, so that secretion rate and hepatic return of bile acids remained constant, despite a wide range of pool sizes. The finding of a normal bile acid synthesis rate in subjects with a small pool size therefore indicated normal feedback regulation of bile acid synthesis. Hourly measurements of biliary lipid output showed a linear relation between bile acid and cholesterol output, with a similar regression line for gallstone and control subjects, but a non-linear relation between bile acid and phospholipid output. Consequently, samples from all subjects were consistently supersaturated with cholesterol at low bile acid outputs, especially during overnight fasting, but not at high bile acid outputs. These findings indicate that hepatic secretion of bile supersaturated with cholesterol is physiological in man at low bile acid outputs, that bile acid pool size is probably determined in part by its recycling frequency, and that cholesterol cholelithiasis in some whites may be due to an underlying extra-hepatic abnormality.

4942 EFFECT OF PHENOBARBITAL IN A CASE OF EXTRAHEPATIC CHOLESTASIS. *(E.)* Metreau, J. M.; Bismuth, H.; Franco, D.; Dhumeaux, D. (Hosp. Henri Mondor, Creteil, France). *Gastroenterology* 68(3): 567-571, 1975.

Treatment of an extrahepatic biliary obstruction in a 44-yr-old woman is described. The woman presented with jaundice and pruritus. For 2 weeks she had complained of nausea, headache, and asthenia and for 10 days had had moderate fever, pruritus, and vomiting. In the preceeding 6-month period she had taken at least 15 different drugs, including opipranol, indomethacin, diazepam, and sulfamethoxydiazine. Probable cholestatic hepatitis was diagnosed. She was discharged on cholestyramine alone, which was stopped after 2 days due to diarrhea. One month later she returned with icterus and pruritus: serum bilirubin was 10.0 mg/100 ml; alkaline phosphatase, 270 IU. Phenobarbital administration was started and slowly increased to 200 mg/day. After 1 week, pruritus and serum bilirubin levels diminished. After 1 month pruritis was reduced, serum bilirubin was 7.0 mg/100 ml. However, the serum alkaline phosphatase remained high at 240 IU. Phenobarbital was stopped. Two months later she suffered a progressive relapse of pruritus and jaundice. Serum bilirubin was 22.0 mg/100 ml; alkaline phosphatase, 660 IU; and total bile acids, 160 µg/ml. Biopsy revealed marked cholestasis and portal sclerosis containing numerous inflammatory cells with few polymorphonuclear leukocytes. Phenobarbital again lowered the pruritus and bilirubinemia and reduced bile acids to 51 µg/ml, but the alkaline phosphatase progressively increased to 1120 IU. Adenocarcinoma was diagnosed and a cephalic pancreatic duodenectomy (Whipple procedure) was sucessfully performed. The phenobarbital-induced decrease in pruritus, serum bilirubin, and bile acids suggests that this drug may be effective in extrahepatic cholestasis, possibly causing enhanced biliary secretion of cholephilic organic anions. It is noted that caution must be taken before interpreting this effect of phenobarbital as relecting the intrahepatic origin of a cholestasis.

4943 ACUTE EMPHYSEMATOUS CHOLECYSTITIS. *(E.)* Abengowe, C. U.; McManamon, P. J. M. (Gen. Hosp., St. John's, Canada). *Can. Med. Assoc. J.* 111 (10):1112-1113, 1974.

A 74-yr-old North American diabetic man with Indian ancestry was hospitalized with a 3-day history of acute, steady, progressive, right upper quadrant abdominal pain associated with nausea and vomiting for 48 hr. Laboratory findings were as follows: leukocyte count 15,000/mm^3, LDH 225 mU/ml, SGOT 76 mU/ml, total bilirubin 1.5 mg/deciliter, serum amylase 40 U/ml, urinary amylase 16 U/ml, fasting blood sugar 197 mg/deciliter, Hb 15 g/deciliter, Hct. 46%, and erythrocyte sedimentation rate 83 mm/hr. A plain film of the abdomen showed an ovoid gas shadow measuring 9 x 5 cm and situated high in the right hypochondrium, which was interpreted as representing gas in the gallbladder. A second plain film made 24 hr later showed the gas involving the gallbladder wall as well. A diagnosis of acute emphysematous cholecystitis was made and a gangrenous distended gallbladder removed at operation. *Clostridium perfringens* was cultured from the gallbladder contents and wall. A review of the literature indicated that acute emphysematous Cholecystitis is more prevalent among male diabetics. Its clinical and laboratory manifestations are similar to those of ordinary acute cholecystitis; diagnosis cannot be made preoperatively without radiologic studies. If acute emphysematous cholecystitis is suspected, it is suggested that intensive antimicrobial therapy and fluid and electrolyte replacement be given prior to early surgical intervention.

4944 EMPTYING OF THE HUMAN GALLBLADDER AND PREDISPOSITION TO GALLSTONE FORMATION. *(E.)* van der Linden, W. (Cent. Hosp., Ostersund, Sweden). *Tijdschr. Gastroenterol.* 17(2):121-128, 1974.

Oral cholecystography performed in patients in whom the gallbladder was visualized without revealing the presence of stones were repeated 30 min after the ingestion of a meal consisting of two egg yolks with sugar to assess the rate of gallbladder emptying. The films were reviewed to estimate gallbladder volume. The patients were separated into two groups, those with a sluggish and those with a vigorous postprandial contraction. In the sluggishly contracting group far more patients were under than over 45 yr of age. In the strong contraction group, more patients were over than under 45 yr of age. Women predominated in the sluggish group while in the other group there was no sex differentiation. During the 13-14 yr between the cholecystographies and this follow-up study, two men in the fibrous

contracting group had been operated on for duodenal ulcer, four patients had been appendectomized, one cholecystectomized and two subjected to gynecological operations. In the sluggishly contracting group there were no gastric operations, four appendectomies, five cholecystectomies, one herniotomy and two gynecological operations. In 12 of the 35 patients who at follow-up could be traced to the sluggish group, cholecystography was repeated and signs of gallstone disease were present in seven. In the fast contraction group, x-ray examination was repeated in nine of the 36 patients who could be traced and gallstone disease was diagnosed in only one. In the sluggishly contracting group, gallstones were found significantly more often when cholecystography was performed some years later. The results of this study show that a sluggish contraction of the gallbladder after an egg yolk meal occurs particularly often in pre-menopausal women. The results further indicate that patients with such gallbladders are especially prone to develop gallstones in later life.

4945 THE CHOLECYSTOKININ TEST: AN ASSESSMENT. *(E.)* Reid, D. R. K.; Rogers, I. M.; Calder, J. F. (R. Infirm., Glasgow, Scotland). *Br. J. Surg.* 62(4):317-319, 1975.

The significance of the cholecystokinin (CCK) test was investigated in 13 patients thought to have x-ray negative gallbladder pain. Duration of the symptoms ranged from 8 months to 10 yr. The patients were told they would be having a cholecystogram (all had one previously) during which they would receive an injection; they were not told the injection might provoke pain. Prone, prone-oblique, and erect films were taken 14 hr after ingestion of six iopanoic acid tablets. Cholecystokinin-pancreozymin was injected over a 1-min period and further films were taken after 2, 4, 6, 8, and 20 min. The injection of 1.5 Ivy U/kg CCK was done into the tubing of an i.v. saline infusion set-up behind the patient so that she was not aware of the moment of injection. The test was considered positive if typical pain was experienced within 10 min. In nine patients, surgery had been indicated clinically. Of these, four were test positive, four test negative, and one was equivocal. Of the four positive reactors, three were relieved by surgery; the fourth had a large functional overlay. Of the four negative reactors, three were relieved by surgery. The remaining patient may have had a small residual stone in the common bile duct after surgery. The patient with equivocal test results was relieved by surgery. Of the four non-operated patients, all were test negative. One was later given a truncal vagotomy and pyloroplasty for duodenal ulcer, but the pains continued. A 14th patient, not included in the series, presented with apparent biliary colic two yr after cholecystectomy gave a positive CCK test. Only three of the patients showed good gallbladder contraction in response to cholecystokinin. It is concluded that a positive test would indicate cholecystectomy while a negative test does not absolve the gallbladder as the source of pain.

4946 ROUX-EN-Y HEPATICJEJUNOSTOMY: A REAPPRAISAL OF ITS INDICATIONS AND RESULTS. *(E.)* Stefanini, P.; Carboni, M.; Patrassi, N.; Basoli, A.; De Bernardinis, G.; Negro, P. (Univ. Rome Med. Sch., Italy). *Ann. Surg.* 181(2):213-219, 1975.

A critical evaluation was made of 131 patients (51 men and 80 women, aged 31-73 yr) submitted to choledocho- or hepaticojejunostomy. The main indications for hepaticojejunostomy were iatrogenic strictures of the common bile duct (60 patients), and choledocholithiasis with markedly dilated duct (41). The overall mortality rate was 4%, principally due to renal hepatic failure, bile peritonitis and bleeding. The complications following hepaticojejunostomy included only one case of biliary fistula which required reoperation. The long-term results of 80 patients available for a follow-up study were as follows: 63 patients (78.7%) were symptom-free at 2-13 yr after surgery; eight patients had brief episodes of cholangitis which responded to antibiotic and corticosteroid treatment; nine patients required reoperation for stricture of anastomosis. These overall results offer a strong argument for hepaticojejunostomy which, compared with choledochoduodenostomy, avoids the hazards of the so-called sump syndrome and of the reflux of enteric contents into the common bile duct. An increased incidence of peptic ulcer disease in the patients submitted to hepaticojejunostomy was not observed. In very high strictures and in reinterventions, anastomosis between left hepatic duct and Roux-en-Y jejunal limb was carried out. The results achieved with this technique in 26 patients were about the same following hepaticojejunostomy. Thus it is concluded that hepaticojejunostomy, as opposed to choledochoduodenostomy, be used in treating obstructions of proximal as well as distal common bile duct. Additionally, hepaticojejunostomy is advised as the most appropriate procedure in patients with choledocholithiasis and markedly dilated duct.

4947 ADENOCARCINOMA OF THE BILE DUCTS. RELATIONSHIP OF ANATOMIC LOCATION TO CLINICAL FEATURES. *(E.)* Ingis, D. A.; Farmer, R. G. (Cleveland Clin., Ohio). *Am. J. Dig. Dis.* 20(3):253-261, 1975.

Case histories of 23 patients with adenocarcinoma of the bile ducts presenting over a 14-yr period were reviewed and a comparison made between carcinoma in the hepatic duct system and carcinomas in the common bile duct. In 14 patients (10 women and four men, aged 38-81 yr) the carcinoma arose in the hepatic duct system. In nine (seven women and two men, aged 44-82 yr) the carcinoma arose in the common bile duct. It was found that age, sex, presenting symptoms, physical examination, and laboratory findings were similar regardless of the location of the carcinoma. Moreover, the location did not influence whether the correct diagnosis was made at the initial operation as carcinomas in both locations, although present, were not found by surgeons at the time of the original operation. Percutaneous cholangiography, operative cholangiography, and endoscopic retrograde cholangiopancreatography were successful in making

the diagnosis in both locations. Other preoperative tests, while showing obstructive jaundice at times, did not aid the surgeon prior to operative intervention. The tumors differed in pathology: hepatic duct carcinomas were often scirrhous; the more distal carcinomas were adenocarcinomas of varied grades. The location influenced the type of surgery. Resection was more common in the distal common bile duct cases. The average survival was less than 2 yr for both locations. In eight hepatic duct carcinoma patients 5-fluorouracil was given p.o. or i.v. weekly following surgery. In two patients cobalt therapy consisting of 2500 - 5000 rads locally to the area of the carcinoma was followed by 5-fluorouracil therapy. In one case, cobalt therapy of 2400 rads alone was given. These measures did not appear to increase the survival of these patients. In the patients with carcinoma of the common bile duct, only one received 5-fluorouracil and he lived only 2 months. Radiation was not used in any of these cases. In view of the poor prognosis for patients with these carcinomas, it is urged that attention be focused on means of earlier diagnosis.

4948 PERCUTANEOUS LIVER BIOPSY IN PATIENTS WITH LARGE BILE DUCT OBSTRUCTION. *(E.)* Morris, J. S.; Gallo, G. A.; Scheuer, P. J.; Sherlock, S. (R. Free Hosp., London, England). *Gastroenterology* 68(4):750-754, 1975.

The safety and diagnostic value of percutaneous liver biopsy were reviewed in 125 patients with large bile duct obstruction. Prior to biopsy, none had palpable gallbladder or suspected cholangitis. In two cases a Vim-Silverman needle was used; in the remainder, a Menghini needle. All patients received 10 mg vitamin K i.m. for 2 days before biopsy. For 24 hr after biopsy, patients were kept in bed, and pulse, temperature and blood pressure were recorded at regular intervals. The biopsy was considered to be diagnostic or suggestive of large bile duct obstruction when most of the following were found: central cholestasis; duct replication; expansion and edema of portal tracts with leukocyte infiltration; and bile infarction. Each patient's record was examined for initial clinical diagnosis, discharge or autopsy diagnosis, complications of liver biopsy, and operative findings. Serum bilirubin levels ranged from 5 to over 20 mg/100 ml, and in 42% jaundice had been present for over 6 months. Percutaneous cholangiography was attempted in 47 patients, and was successful in 38. Of the 125 patients, eight developed complications: biliary peritonitis (one); severe pain in the upper right abdomen (three); pyrexia and chills (two); pleural effusion and blood-stained ascites (one each). There was no apparent relation between development of complications and the depth of jaundice or the anatomical abnormality. Needle biopsy failed in two patients. In 103 cases, the initial biopsy showed changes compatible with large duct obstruction, in 16 the sole abnormality was cholestasis, and in six, results were unhelpful or misleading. Of 38 patients in whom the ducts were successfully punctured, 32 showed changes associated with large duct obstruction, and six cholestasis. It is concluded that needle biopsy is useful and safe to perform in patients with large duct obstruction; however, a careful prebiopsy work-up and close observation for 24 hr after biopsy are essential.

4949 CHOLECYSTECTOMY PEER REVIEW: MEASUREMENT OF FOUR VARIABLES. *(E.)* Mitchell, J. H.; Hardacre, J. M.; Wenzel, F. J.; Lohrenz, F. N. (Marshfield Clin., Wisconsin). *Med. Care* 13(5):409-416, 1975.

Patients undergoing uncomplicated cholecystectomy (222) were evaluated by both process and outcome methods, and their satisfaction and duration of postoperative stay were measured. Criteria for process analysis were: indications for admission; laboratory tests; length of postoperative stay; indications for discharge; and other components for a comprehensive medical record. Of the 222, 80 at random were used as the basis for this analysis. Outcome was divided into five types: asymptomatic and back-to-normal activities; symptomatic and back-to-normal activities; not back-to-work or normal activities; bedridden; and dead. Information was gathered from the medical records and patients answered a questionnaire to determine outcome status and satisfaction. After the analyses were completed, the patients were divided into those with bile duct exploration and those without. Each group was subdivided according to surgeon, and mean postoperative stay for each surgeon's patients calculated for each variable. The major omission found by process review was the absence of preoperative serum amylase test in eight of 18 patients with acute cholecystitis. In one instance there was no record of urinalysis or hematocrit. All had abnormal gallbladders documented by pathological reports. Outcome analysis results were within predicted expectations in four categories. In the symptomatic back-to-work group, however, there was 31% of the patients, in contrast to the predicted 13%. There were no statistical differences in length of postoperative stay among the surgeons of the bile duct exploration group. In contrast, the group given no such exploration showed a marked difference in stay among their surgeons. One surgeon kept patients an average of two days longer than another in the group. Of all the patients, 95% were satisfied with their care and 90% felt the length of their postoperative stay was appropriate. It is concluded that process outcome and patient satisfaction were satisfactory, and length of postoperative stay may be shortened.

4950 BILE ACID PATTERN OF GALLBLADDER AND HEPATIC BILE IN PATIENTS WITH AND WITHOUT CHOLESTEROL GALLSTONES. *(E.)* Arnesjo, B.; Bodvall, B.; Stahl, E. (Dep. Surg., Univ. Lund, Sweden). *Acta Chir. Scand.* 141(2):135-138, 1975.

Gallbladder bile acid pattern was compared in 18 patients with cholelithiasis and 13 with duodenal ulcer disease. Both gallbladder and hepatic bile specimens were taken after an overnight fast during cholecystectomy or selective proximal vagotomy.

Bile acids were immediately extracted with methanol acetone and separated by thin layer chromatography. Molar concentrations were determined, as well as the ratios of molar concentrations of glycine:taurine conjugated acids, trihydroxy:dihydroxy acids, cholate:deoxycholate, cholate:chenodeoxycholate, cholate plus deoxycholate:chenodeoxycholate and chenodeoxycholate:deoxycholate. In each case, hepatic bile molar ratios were divided by gallbladder bile molar ratios. The individual hepatic to gallbladder bile acid molar ratios were then compared with those of the control duodenal ulcer patients. The total bile acid concentration of the gallbladder bile in the controls (164.7) was higher than that of the gallstone group (122.9). There were no differences between the two groups in bile acid patterns, excepting a lower trihydroxy:dihydroxy ratio in the gallstone group. Individual hepatic to gallbladder relative molar ratios in the gallstone group had elevated trihydroxy:dihydroxy (1.35) and cholate:deoxycholate (1.47) ratios. It is concluded that gallbladder bile from gallstone patients has a diminished trihydroxy:dihydroxy molar ratio.

4951 GALL-STONES AND ORAL CONTRACEPTIVES. *(E.)* Howat, J. M. T.; Jones, C. B.; Schofield, P. F. (Park Hosp., Manchester, England). *J. Int. Med. Res.* 3(2):59-62, 1975.

Female patients (100) were studied to determine whether oral contraceptives increased risk of gallstone formation. Of these, 50 presented for surgery with gallstones, and 50 were controls admitted for minor surgery or as a result of trauma. A record was kept of those who had taken oral contraceptives for over six months at any time prior to admission. The number of pregnancies and miscarriages were recorded so the two groups could be matched for parity. Of the 50 gallstone patients, 35 (70%) had used contraceptives for at least six months, as compared to 14 (28%) of the controls, a highly significant difference. Parity and marital status did not differ between the groups. It is concluded that there is a markedly higher oral contraceptive use in gallstone patients than in normal controls.

4952 STUDIES ON BILE SALT DECONJUGATION FOLLOWING SMALL BOWEL BYPASS PROCEDURES. *(E.)* Wise, L.; Margraf, H.; Stein, T. (Washington Univ. Sch. Med., St. Louis, Mo.). *Ann. Surg.* 181(4):397-401, 1975.

The amount of bile salt deconjugation and malabsorption following small bowel bypass was studied, and the role of the glycine-1-^{14}C-cholic acid (^{14}C-GCA) test in evaluating the intestinal adaptive response and bypass adequacy was assessed. Patients (26) with morbid obesity had 35 cm of proximal jejunum anastomosed end-to-end to 10 cm of terminal ileum. Glycine-1-^{14}C-cholate was given p.o. and the specific activity of the expired $^{14}CO_2$ was determined to serve as an index of bacterial deconjugation. The ^{14}C content of 48-hr stool specimens following ingestion of the label served as a malabsorption index. These studies were performed a week prior to surgery and also 3-4 days, 8-10 days, and 5-11 months after surgery. During the postoperative period, $^{14}CO_2$ excretion was no higher than 0.1% dose/mM CO_2/kg, even after 6 hr. In the immediate postoperative period (during antibiotic therapy) there was no measurable $^{14}CO_2$ excretion, but after 8-10 days there was a significant increase. High excretion started within 2 hr (0.51 x 0.12) and persisted for about 8 hr. At 6 hr the increase was 20%. After 5-10 months there was a significant 10% increase in early CO_2 output (0.37 x 0.05 at 4 hr) for six hr. Mean ^{14}C respiratory loss during the first 6 hr was 2.2%, in the 8-10 day group 25.4%, and in the 5-11 month group 15.9%. The percentage of loss was increased significantly in both postoperative groups over the preoperative group and in the early as opposed to the late postoperative group. Mean fecal ^{14}C excretion was 6.6% of the preoperative dose, 50.9% in the early postoperative period, and 21.2% in the late postoperative period, indicating a decrease and compensatory increase in bile salt absorption. It is concluded that the ^{14}C-GCA test may be used to follow the adaptive response following bypass surgery.

4953 SCLEROSING CHOLANGITIS: SURGICAL SIGNIFICANCE. *(E.)* Grodsinsky, C.; Block, M. A.; Brush, B. E. (Henry Ford Hosp., Detroit, Mich.). *Henry Ford Hosp. Med. J.* 22(4):211-216, 1974.

A review is presented of six cases of possible sclerosing cholangitis. Criteria for inclusion of a case were: absence of previous biliary tract surgery and of gallstones; diffuse involvement of the extrahepatic ducts; and exclusion of cholangiocarcinoma. All had been treated for extrahepatic lesions; in two the intraoperative findings were similar. Absence of postmortem examinations made confirmation of intraoperative diagnosis impossible. At least three patterns of anatomic involvement were observed. The first, the segmental sclerosing type, results in recurrent bouts of cholangitis which may be relieved by surgical bypass of the obstruction. Stenosis is limited to a segment, usually distal, of the extrahepatic ducts. Prognosis is good. The diffuse sclerosing type, in contrast, is usually associated with chronic ulcerative colitis, and involves the entire biliary tract, intra- and extrahepatic. The third type, primary fibrosing pericholedochitis, involves the extrahepatic tract and is difficult to distinguish from other lesions causing chronic inflammation in this region. Subsequent clinical course after surgery must be reviewed to establish diagnosis. It is suggested that all possible mechanical methods of relief through surgery should be attempted, and steroids administered as an alternative where the first is ineffective or impossible.

4954 BILIOBILIARY FISTULA. A TRAP IN THE SURGERY OF CHOLELITHIASIS. *(E.)* Corlette, M. B.; Bismuth, H. (Paul Brousse Hosp., Villejuif, France). *Arch. Surg.* 110(4):377-383, 1975.

Case studies are presented for 24 patients (5 men and 19 women, average age 60.2 yr) treated for calculous biliobiliary fistula. Jaundice was the most common

symptom, usually sudden in onset and associated with pain. The duration of biliary symptoms ranged from 15 days to 20 yr. Marked weight loss occurred in six. Bilirubin ranged from 2.5-14.8 mg/100 ml; alkaline phosphatase, 35-600 IU. At operation a constant and important finding was marked adhesions in the right upper quadrant fusing the adjacent organs into a chronic inflammatory mass. The gallbladder was shrunken and thickly scarred in all but two patients, in whom it was necrotic and removed in fragments. In no case was there a distinguishable line of demarcation between gallbladder and main bile duct, and the latter could not be readily dissected from the gallbaldder. In 18 cases the location in which an abnormal communication occurred between gallbaldder and main bile duct was between the Hartmann pouch or the ampulla of Vater and the hepatic duct. In six cases the gallbladder neck was continuous with the main bile duct, and a large direct communication existed between the gallbladder and the common duct in the trajectory of the cystic duct. A stone larger than 1.5 cm in diameter was found in 21 cases. In 11 cases a huge stone was astride the fistula, partly within the gallbladder and projecting into and obstructing the main bile duct. Treatment consisted of removal of stones and partial cholecystectomy down to the fistulized zone, leaving a remnant of gallbladder tissue about the abnormal communication. A T-tube was inserted through the fistula and the duct closed about it in 21 patients. In three cases an hepaticojejunostomy was performed. Postoperative complications included septis (two), partial transitory bile fistula, and self-limited episode of gastrointestinal tract bleeding (one each). One patient became jaundiced 4 months after closure of the fistula and at reexploration had developed a carcinoma obstructing the hepatic duct. It is emphasized that no attempt should be made to dissect the main bile duct from the gallbladder due to great danger to the duct. By opening the gallbladder peripherally and obtaining a cholangiogram, such an abnormal communication can be diagnosed. It is best treated by leaving a small collar of gallbladder tissue about the fistula and using this tissue to close the main duct over a T-tube.

4955 STUDIES IN CHOLESTASIS OF PREGNANCY. I. CLINICAL ASPECTS AND LIVER FUNCTION TESTS. *(E.)* Johnson, P.; Samsioe, G.; Gustafson, A. (Sahlgrenska Hosp., Univ. Goteberg, Sweden). *Acta Obstet. Gynecol. Scand.* 54(1):77-84, 1975.

Consecutive cases of pruritus in pregnancy (59) were investigated; 57 were without obvious dermatological causes and were further studied for clinical features related to the occurrence of lipoprotein X (LP-X) in serum and to liver function tests. The onset of pruritus due to cholestasis was during the third trimester in 65% of the patients. The occurrence of new cases was highest in the sixth and eighth months of pregnancy. Emesis was more common among patients with cholestatis (75%) than among controls (58%). Gallbladder disease was more frequent in pregnant women with pruritus than in controls. Food and drug idiosyncracy was more common in cholestasis of pregnancy and included difficulties with penicillins, sulfonamides, strawberries, fish, and oranges. There was no significant difference in age between cholestasis of pregnancy patients and controls. The time of delivery was normal in both groups. The amount of uterine bleeding during delivery was normal in each group, as was the patient distribution among A, B, O, and Rh blood groups. The newborn infant of the mother who had suffered from pruritus during pregnancy was not afflicted with a higher perinatal mortality nor was the incidence of immaturity and dysmaturity higher than in controls. Pregnant women with pruritus due to cholestasis showed wide ranges of values for liver function tests, serum bilirubin, alkaline phosphatase, SGOT and SGPT. In only three cases was the serum bilirubin elevated, while only a minor portion of the values of alkaline phosphatase were within normal limits. SGOT and SGPT followed a pattern similar to alkaline phosphatase, increasing in relation to the length of gestation. The cholestatic cause of the pruritus was verified in the 57 patients by the presence of LP-X in serum as determined by the immunological technique. LP-X was frequently present in serum together with normal alkaline phosphatase, SGOT, and SGPT and was particularly notable in cases in whom the onset of pruritus was in the second trimester. The finding of prolonged and severe emesis early in pregnancy suggests that the basic and possibly metabolic disturbance causing cholestasis is present throughout pregnancy, even though the onset of pruritus in cholestasis of pregnancy is most frequent in the last trimester. It is suggested that the modified immunological test for the detection of LP-X in serum may be a useful diagnostic tool in the diagnosis of cholestasis of pregnancy.

4956 A RARE CASE OF CYSTIC DILATATION OF THE MAIN BILE DUCT ("CHOLEDOCHOCELE"). *(It.)* Pelizzo, M. R.; Stella, G. D.; Masutti, A. (Inst. Surg, Pathol., Univ. Padua, Italy). *Chir. Ital.* 26(5): 488-494, 1974.

4957 CASES OF EXTRAHEPATIC PORTAL VEIN OBSTRUCTION ACCOMPANIED BY BILIARY TRACT DISEASE. *(Jpn.)* Akita, H.; Sakoda, K.; Kawada, T.; Arima, Y.; Katsuki, T. (Dep. Surg.II, Kagoshima Univ. Japan). *Acta Med. (Fukuoka)* 44(2):104-105, 1974.

4958 THE KINETICS OF THE GALLBLADDER IN CONGENITAL ANOMALIES. *(Ger.)* Stefanow, W.; Schilkow, P. (Dr. P. Kolarow Munic. Hosp., Sofia, Bulgaria). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 121(2):213-216, 1974.

4959 RE-OPERATION ON THE BILE DUCTS: 56 CASES IN TWO YEARS. *(Fr.)* Doutre, L. P.; Perissat, J.; Gouffrant, J. M.; Bobois, J. P. (Surg. Clin. A, Bordeaux, France). *J. Chir. (Paris)* 107(2): 131-138, 1974.

4960 THE EPIDEMIOLOGY OF CHOLELITHIASIS. *(Ger.)* Back, P. (Med. Clin., Univ. Freiburg, Germany). *Leber Magen Darm* 4(1):13-16, 1974.

4961 CONGENITAL DILATATION OF THE INTRAHEPATIC BILIARY TRACT. CAROLI'S DISEASE. *(Fr.)* Juvara, I.; Radulesco, D.; Pacescu, E. (Dr. I. Cantacuzino Hosp., Bucharest, Roumania). *Lyon Chir.* 71(1):56-57, 1975.

4962 INDICATIONS FOR AND RESULTS OF SURGICAL TREATMENT OF CHOLELITHIASIS. *(Ger.)* Brandt, P.; Ungeheuer, E. (Northwest Hosp., Frankfurt/Main, Germany). *Leber Magen Darm* 4(2):75-79, 1974.

4963 BENIGN TUMORS OF THE GALLBLADDER. *(Ger.)* Wenzl, M. (Sophiensp., Vienna, Austria). *Leber Magen Darm* 4(2):79-80, 1974.

4964 TORSION OF THE GALLBLADDER. *(Ger.)* Schwesinger, G.; Ventz, M. (Inst. Pathol., Ernst Moritz Arndt Univ., Griefswald, Germany). *Z. Gesamte. Hyg.* 30(4):162-163, 1975.

4965 BILIARY AMYLASE ACTIVITY. *(Fr.)* Paris, J.; Salembier, Y.; Gerard, A.; Roger, J. (Reg. Hosp. Cent., Lille, France). *Tijdschr. Gastroenterol.* 17(3):192-204, 1974.

4966 SURGICAL REPAIR OF TRAUMATIC LESIONS OF THE BILIARY TRACT (14 CASES). *(Fr.)* Ampe, J.; Thery, D.; Langeron, P. (St. Philibert Hosp., Lille, France). *Tijdschr. Gastroenterol.* 17(3):177-191, 1974.

4967 EFFECT OF PHARMACOLOGICAL VAGOTOMY ON THE CHOLERETIC FUNCTION OF THE LIVER. *(Pol.)* Krajka, K.; Anisimowicz, L. (Acad. Med., Gdansk, Poland). *Przegl. Lek.* 31(2):311-316, 1974.

4968 ACCESSORY INTRAHEPATIC GALLBLADDER. *(Pol.)* Wolnicki, S.; Swadzba, S.; Borgiel, W. (Munic. Hosp. 2, Bielsko-Biala, Poland). *Pol. Przegl. Radiol.* 38(5):629-633, 1974.

4969 CHOLELITHIASIS IN PATIENTS OVER 65 YEARS OLD. *(Pol.)* Pioch, E. (Acad. Med., Poznan, Poland). *Pol. Przegl. Chir.* 46(16):1309-1314, 1974.

4970 GALLBALDDER AGENESIS AND ITS SURGICAL TREATMENT. *(Cz.)* Safranek, V. (Mil. Hosp., Plzen, Czechoslovakia). *Cz. Gastroenterol. Vyz.* 28 (7):483-485, 1974.

4971 AGENESIS OF THE GALLBLADDER AND THE COMMON BILE DUCT. *(Cz.)* Bocz, M. (Reg. Public Health Cent., Nove Zamky, Czechoslovakia). *Rozhl. Chir.* 53(7):460-463, 1974.

4972 IMPORTANCE OF DRAINAGE OF THE ABDOMINAL CAVITY AFTER SIMPLE CHOLECYSTECTOMY. *(Cz.)* Kouba, R. (Reg. Public Health Cent., Karlovy Vary, Czechoslovakia. *Rozhl. Chir.* 53(7):464-469, 1974.

4973 MANOMETRY OF PERFUSION FLUID IN THE CYSTIC DUCT AND ODDI'S SPHINCTER. *(Rus.)* Duberman, L. B. (Vologda Reg. Hosp., USSR). *Khirurgiia (Mosk.)* (3):93-99, 1974.

4974 DIAGNOSIS OF INFLAMMATION OF THE GALLBLADDER AND BILIARY DUCTS. *(Rus.)* Koshevskaya, L. I.; Bulkina, G. I.; Bondarenko, G. V. (Vinnitsa Med. Inst., USSR). *Vrach. Delo* 616(2):53-55, 1975.

4975 DISORDERS AND TREATMENT OF AMINO ACID METABOLISM IN PATIENTS WITH CHRONIC DISEASES OF THE BILIARY TRACT. *(Rus.)* Krylova, T. V. (Lvov Med. Inst., USSR). *Vrach. Delo* 616(2):48-50, 1975.

4976 THE CLINICAL COURSE OF CALCULOUS CHOLECYSTITIS COMPLICATED BY OBSTRUCTIVE JAUNDICE IN OLD PATIENTS. *(Rus.)* Ashrefov, A. A. (N. Narimanov Azerbaidzhan Med., Inst., USSR). *Sov. Med.* (9):68-72, 1974.

4977 RECANALIZATION OF TUMOR-OCCLUDED MAIN BILE DUCTS. *(Rus.)* Shubin, B. M. (P. A. Gertsen Res. Inst. Oncol., Moscow, USSR). *Khirurgiia (Mosk.)* (3):89-93, 1974.

4978 CHOLESTEROSIS OF THE GALLBLADDER IN ELDERLY PATIENTS. *(Rus.)* Melnik, L. A. (Crimean Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):53-57, 1974.

4979 SUBCUTANEOUS AND PENETRATING INJURIES OF THE GALLBLADDER. *(Rus.)* Korenev, N. N.; Shumanova, T. I.; Ustimenko, Y. M. (I. V. Skifosovskii Res. Inst. First Aid, Moscow, USSR). *Khirurgiia (Mosk.)* (3):49-53, 1974.

4980 CHANGES IN BLOOD COAGULABILITY IN HEPATORENAL INSUFFICIENCY. *(Rus.)* Sergienko, V. S. (Cent. Inst. Grad. Med. Studies, Moscow, USSR). *Khirurgiia (Mosk.)* (3):40-44, 1974.

4981 THE MANAGEMENT OF PATIENTS WITH ACUTE CHOLECYSTITIS. *(Rus.)* Burlachenko, V. P. (Inst. Transplant Org. Tissues, Moscow, USSR). *Khirurgiia (Mosk.)* (3):22-25, 1974.

4982 ACALCULOUS CHOLECYSTITIS. *(Rus.)* Lidsky, A. T.; Lisiyenko, V. M.; Shveitser, E. A. (no affil.). *Khirurgiia (Mosk.)* (3):18-21, 1974.

4983 EXTERNAL DRAINAGE OF THE BILE DUCTS. *(Rus.)* Vinogradov, V. V.; Venkatadri, G.; Pryadkin, V. V. (Dep. Surg., P. Lumumba Friendship Univ., Moscow, USSR). *Khirurgiia (Mosk.)* (3):8-13, 1974.

4984 INDICATIONS FOR AND CHOICE OF METHOD OF INTERNAL BILE DRAINAGE IN CHRONIC CHOLECYSTITIS. *(Rus.)* Mayat, V. S.; Malinovsky, N. N.; Ovchininsky, N. N.; Dasayev, A. N.; Rubashnaya, I. E. (no affil.). *Khirurgiia (Mosk.)* (3):3-8, 1974.

4985 DECOMPRESSION DRAINAGE OF CYSTIC BILE DUCT IN SURGERY OF NON-TUMOR DISEASES OF THE BILIARY TRACT. *(Rus.)* Nosov, I. S. (Surg. Hosp., Plovdiv Univ., Bulgaria). *Khirurgiia (Mosk.)* (3): 84-88, 1974.

4986 ERYTHROMYCIN THERAPY OF CHILDREN WITH BOTKIN'S DISEASE AND INVOLVEMENT OF THE BILE DUCTS. *(Rus.)* Lupanova, R. I. (Leningrad Med. Pediatr. Inst., USSR). *Vopr. Okhr. Materin. Det.* 18(12):9-13, 1973.

4987 STUDY OF THE PORTAL CIRCULATION IN OBSTRUCTIVE JAUNDICE. *(Rus.)* Krakovsky, A. I.; Ivannikov, N. F. (Surg. Hosp., Tiumen Med. Inst., USSR). *Khirurgiia (Mosk.)* (9):81-85, 1974.

4988 REOPERATIONS ON THE BILIARY TRACT. *(Rus.)* Sergeev, S. I.; Tashkinov, V. T. (Khabarovsk Med. Inst., USSR). *Khirurgiia (Mosk.)* (9):75-81, 1974.

4989 CHOLEDOCHODUODENOSTOMY IN CHOLELITHIASIS. *(Rus.)* Korolev, B. A.; Mironov, O. N.; Gagushin, V. A. (Gorkii Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):13-18, 1974.

4990 SURGERY OF BILE DUCT OBSTRUCTION AT THE PORTA HEPATICA. *(Rus.)* Galperin, E. I.; Burlachenko, V. P. (Inst. Transplant. Org. Tissues), Moscow, USSR). *Khirurgiia (Mosk.)* (9):71-75, 1974.

4991 OBSTRUCTIVE JAUNDICE FROM FREE SUTURES IN THE BILIARY DUCT AS A COMPLICATION OF GASTRODUODENAL RESECTION FOR ULCER AND OF SIMPLE CHOLECYSTECTOMY FOR LITHIASIS. TWO CASE REPORTS. *(It.)* Fani, C.; Puntillo, E.; Bressan, G. C. (Pio Inst. S. Spirido Hosp. Inst., Rome, Italy). *Policlinico Sez. Chir.* 80(5):253-266, 1973.

4992 LATE FILLING OF THE GALLBLADDER. *(Ger.)* Weisel, M.; Maassen, G. (Munic. Hosp., Berlin-Kopenick, Germany). *Radiol. Diagn. (Berl.)* 15(1):9-12, 1974.

4993 THE REDUCTION OF GLUCOSE IN VASCULAR AND CHOLEDOCHAL BILE IN HUMANS. *(Fr.)* Paris, J.; Gerard, A.; Salembier, Y.; Samso, G. (no affil.). *Tijdschr. Gastroenterol.* 17(1):40-50, 1974.

4994 A CHOLECYSTOCOLIC FISTULA CAUSED BY PERFORATION OF A GALLBLADDER CANCER. *(Fr.)* Godquin, B.; Favre, R.; Blanchard, J.; Boutovitch, N. (no affil.). *Acta Gastroenterol. Belg.* 37(4/5): 236-242, 1974.

4995 D-PENICILLAMINE IN RECONSTRUCTIVE SURGERY OF THE BILIARY TRACT: BILIODIGESTIVE ANASTOMOSES. *(Ger.)* Henrich, M. (Cent. Surg., Univ. Frankfurt/Main, Germany). *Munch. Med. Wochenschr.* 116(50):2213-2214, 1974.

4996 GALLSTONE ILEUS. *(Ger.)* Togel, H. (Dist. Hosp., Giessen, Germany). *Munch. Med. Wochenschr.* 116(50):2195-2198, 1974.

4997 CHOLELITHIASIS IN PRIMARY HYPERPARATHYROIDISM. *(Ger.)* Funk, C.; Ammann, R.; Binswanger, U.; Mayor, G.; Bihrer, R.; Clavadetscher, P.; Fumagalli, I.; Leemann, A.; Seiler, P.; Stuby, K. (Urol. Clin., Univ. Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 104(30):1060-1064, 1974.

4998 MORPHOLOGY OF GALLSTONES. *(Ger.)* Wolpers, C. (Inst. Phys. Med., Univ. Munster, Germany). *Leber Magen Darm* 4(2):43-57, 1974.

4999 ADVANCES IN THE DIAGNOSIS OF CHOLELITHIASIS. *(Ger.)* Meyer-Burg, J. (Steglitz Clin., Free Univ. Berlin, Germany). *Leber Magen Darm* 4(2):58-64, 1974.

5000 MECHANICAL FACTORS IN GALLSTONE FORMATION. *(Ger.)* Stauber, R. (Leoben Dist. Hosp., Germany). *Leber Magen Darm* 4(1):27-31, 1974.

5001 BLOOD LIPIDS AND CHOLELITHIASIS. *(Ger.)* Heschi, R.; Pristautz, H. (Med. Clin., Univ. Graz, Austria). *Leber Magen Darm* 4(1):25-26, 1974.

5002 SOME ASPECTS OF BILE SALT TURNOVER IN PATIENTS WITH GALLSTONES. *(Ger.)* Brunner, H.; Horak, W. (Med. Clin. II, Univ. Vienna, Austria). *Leber Magen Darm* 4(1):22-24, 1974.

5003 THE PATHOPHYSIOLOGICAL BASIS OF GALLSTONE FORMATION. *(Ger.)* Paumgartner, G. (Inst. Clin. Pharmacol., Univ. Bern, Switzerland). *Leber Magen Darm* 4(1):17-21, 1974.

5004 THE PATHOPHYSIOLOGICAL BASIS OF GALLSTONE DISSOLUTION. *(Ger.)* Erb, W. (Cent. Intern. Med., Univ. Franfurt, Germany). *Leber Magen Darm* 4(2):72-74, 1974.

5005 CONGENITAL AGENESIS OF THE GALL-BLADDER. *(It.)* Benintendi, V.; Salmaso, G. C.; Eccher, C.; Fontanin, O. (Civil Hosp., Padua, Italy). *Acta Chir. Ital.* 30(2):209-220, 1974.

5006 BILIO-BILIARY FISTULA OF THE GALLBLADDER INFUNDIBULUM AND THE RIGHT HEPATIC DUCT. (4 PERSONAL CASES). *(It.)* Battistini, C.; Sianesi, M. (Inst. Spec. Surg. Pathol. Clin. Propedeut., Univ. Parma, Italy). *Acta Chir. Ital.* 30(2):181-192, 1974.

5007 CARCINOMA OF THE BILE DUCTS. *(Ger.)* Moschinski, D.; Sailer, R.; Jacobs, G. (Surg. Clin., Univ. Dusseldorf, Germany). *Bruns. Beitr. Chir.* 221(8):592-598, 1974.

5008 SPECIFIC AND NON-SPECIFIC LYMPH NODE ENLARGEMENT DUE TO COMMON BILE DUCT COMPRESSION. *(Ger.)* Jakab, F.; Gonci, L. (Semmelweis Med. Univ., Budapest, Hungary). *Bruns. Beitr. Chir.* 221 (8):599-604, 1974.

5009 HEPATIC CONJUGATION OF CHENODEOXYCHOLIC AND CHOLIC ACIDS. *(Ger.)* von Bergmann, K.; Schultheiss, H. R.; Paumgartner, G.; Preisig, R. (Inst. Clin. Pharmacol., Univ. Bern, Switzerland). *Schweiz. Med. Wochenschr.* 105(13):413-415, 1975.

5010 CHOLELITHIASIS AND ITS COMPLICATIONS. *(Rus.)* Shabanov, A. N.; Mikeladze, K. D.; Urazov, K. B. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* 616(5):115-120, 1974.

5011 FOLLOW-UP RESULTS OF CHOLECYSTOSTOMY FOR ACUTE CHOLECYSTITIS AND CHOLANGITIS CAUSED BY OPISTHORCHIASIS. *(Rus.)* Ziganshin, R. V.; Tretiakov, A. M. (Tyumen Med. Inst., USSR). *Khirurgiia (Mosk.)* 616(9):85-88, 1974.

5012 CHOLANGIOGRAPHY AND MANOMETRY AFTER OPERATIONS ON THE BILE DUCTS. *(Rus.)* Obukhov, V. A.; Travkin, Iu. A.; Gorbunov, V. V. (Inst. Med. Stomatol., Moscow, USSR). *Khirurgiia (Mosk.)* 616(3): 66-68, 1974.

See also, 4194, 4197, 4213, 4250, 4256, 4257, 4267, 4272, 4273, 4274, 4275, 4276, 4277, 4278, 4279, 4280, 4281, 4286, 4287, 4288, 4295, 4296, 4570, 4571, 4609, 4744, 4838, 4916, 4941, 5042, 5057, 5068, 5193.

GENERAL

5013 AMPICILLIN-ASSOCIATED DIARRHEA--A PROSPECTIVE STUDY. *(E.)* Tedesco, F. J. (Washington Univ. Sch. Med., St. Louis, Missouri). *Am. J. Dig. Dis.* 20(4):295-297, 1975.

A prospective study of 200 consecutive patients (131 women and 69 men, aged 15-91 yr) given ampicillin for various reasons showed that bowel habits changed in 32 (16%) and that 9 of the 32 (4.5%) had diarrhea. The incidence of change in bowel habits increased with total dosage and the change occurred with equal frequency after p.o. and i.v. administration of ampicillin. Proctoscopic examination of 17 patients with changed bowel habits showed edema in all patients with more than 5 bowel movements/day and erythema in 1/2 the patients. However, no case of pseudomembrane colitis was found. A comparable study of 200 patients treated with clindamycin had revealed a 21% incidence of diarrhea and a 10% incidence of pseudomembrane colitis.

5014 IMMUNOFLUORESCENCE IN DUODENAL MUCOSA OF CHILDREN WITH ACUTE ENTERITIS DUE TO A NEW VIRUS. *(E.)* Davidson, G. P.; Goller, I.; Bishop, R. F.; Townely, R. R. W.; Holmes, I. H.; Ruck, B. J. (R. Child. Hosp., Melbourne, Australia). *J. Clin. Pathol.* 28(4):263-266, 1975.

Electron microscopy of duodenal mucosa from 8 children with acute nonbacterial enteritis had shown duovirus particles in epithelial cells. Indirect immunofluorescent techniques were applied to the same tissue and showed virus antigen localized in the cytoplasm of epithelial cells of the villi. Fluorescence occurred with fluorescein-labeled antisera to human IgM but not with similarly labeled antisera to IgG or IgA. Specific IgM antibody was present in sera from infected patients as early as 2 days after the onset of symptoms. Virus particles from different patients appeared to share a common antigen since the particles fluoresced when incubated with heterologus sera. These results support the belief that duovirus was the cause of acute enteritis in the children studied.

5015 GASTROINTESTINAL BLEEDING: TREATMENT WITH INTRA-ARTERIAL VASOPRESSIN. *(E.)* Geronilla, D. R.; Sampliner, J. E.; Lerner, A. (Mt. Sinai Hosp., Cleveland, Ohio). *Am. Surg.* 41(5):321-326, 1975.

The use of selective intra-arterial vasopressin in the treatment of 17 cases (12 male, five female) of gastrointestinal hemorrhage is discussed. Vasopressin was administered by continuous pump infusion through either the celiac or the superior mesenteric

arteries. Of 17 patients, 16 had bleeding coming from above the ligament of Treitz and only one patient had lower gastrointestinal bleeding. Vasopressin was infused with a starting dose of 0.4 U/min for 20-30 min in the celiac artery, then reduced to 0.2 U/min. Superior mesenteric artery infusion was started with 0.2 U/min. Vasopressin was gradually tapered after cessation of bleeding and catheter removal was accomplished in less than 3 days. The treatment stopped the gastrointestinal hemorrhage in 13 of 17 patients (76.5%). No major complications occurred. Despite the early successes of this treatment, its therapeutic indications are still not fully established. It is suggested, however, that vasopressin has a use in stabilizing the condition of the bleeding patient, providing time for full diagnostic evaluation, and converting an emergency surgical procedure to an elective one.

5016 CHEMICALLY DEFINED DIET IN THE TREATMENT OF KWASHIORKOR. *(E.)* Habte, D.; Persson, B.; Sterky, G. (Dep. Pediatr., Haile Selassie I Univ., Addis Ababa, Ethiopia). *Acta Pediatr. Scand.* 64(2): 267-272, 1975.

A synthetic feeding mixture with an almost exclusively monomolecular composition was tested in children with kwashiorkor. Of 21 children, 13 received the synthetic food and eight received a standard diet with cow milk protein concentrate. Feeds were given every third hr at 100 mg/kg/day in the beginning, usually *via* nasogastric tube, for the first few days. Tea with sugar was given to all patients on the first day, half strength nutrient on the second day and full strength on the third day. Rectal temperature was taken before each feed and 1, 2, and 3 hr after. Venous blood was drawn on the second day of admission, every second day for the first week, and weekly thereafter, to be analyzed for blood urea nitrogen, glucose, and albumin. Hemoglobin was determined from capillary blood. There were six deaths, four in the synthetic and two in the standard-fed groups. Four were due to dehydration from diarrhea, one from hypoglycemia and one from congestive heart failure. The mortality corresponded to that of the in-patients of the hospital. The synthetic-fed group took the food from the bottle by the second or third day, showing rapid increase in appetite, while the standard-fed group had to be tube-fed for about a week. The synthetic-fed group improved more quickly than the standard-fed group, the groups taking 7.1 and 14.1 days, resp., to attain minimum weight. Subsequent weight gain was the same in both groups. Frequency of vomiting was significantly higher in the standard-fed group. There was a rise in rectal temperature in most of the patients, but was more pronounced and significant during the first half of the week in the synthetic-fed group. Albumin and blood urea nitrogen showed a progressive rise in both groups and became significant in the standard-fed group on day eight and the synthetic-fed group on day 15. There was also a significant increase in hemoglobin concentration during the first 3 weeks in the synthetic-fed group only. It is concluded that a completely chemically-defined diet can successfully reverse acute kwashiorkor, with minimum weight reached sooner and with less protein than the standard diet.

5017 IMPORTANCE OF MULTIPLE EPISODES OF HYPOXIA OR COLD STRESS ON THE DEVELOPMENT OF ENTEROCOLITIS IN AN ANIMAL MODEL. *(E.)* Barlow, B.; Santulli, T. V. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Surgery* 77(5):687-690, 1975.

The effects of cold stress or of the number of episodes of cold stress or hypoxia on the development of acute necrotizing enterocolitis was investigated in the rat. Split litters of newborn Sprague-Dawley rats were used for breast-fed and formula-fed groups for each experimental condition. Cold stress by refrigeration for 5 min at 7 C was administered once daily for 1, 2, or 3 days for some groups. Hypoxia by enclosure of the animals in a plastic bag for 3-5 min was administered once daily for 1, 2, 3, or 4 days to other groups. All rats were given an oral innoculum of *Klebsiella* bacteria at birth. Fecal and blood cultures and pathological examination of the gut were performed on all rats. Diseased rats were killed when in a terminal state, and healthy survivors when 7 days old. None of the breast-fed litter mates in the cold or hypoxia stressed groups showed any pathological changes. Stool cultures showed rare gram-negative colonies at 7 days. The formula-fed rats showed development of necrotizing enterocolitis in direct relation to the number of stress episodes. The disease occurred at 2-5 days of age in 60% after one, and 100% after two or more cold stress episodes. Hypoxia caused the disease in 20% after one stress, 60% after two, 80% after three, and 100% after four. Fecal and blood cultures all showed heavy *Klebsiella* growth. It is concluded that a critical amount of enteric ischemia, as caused by various forms of stress, is required to initiate enterocolitis, and that breast milk protects against the disease by control of bacterial growth.

5018 USE OF AN ORAL ELEMENTAL DIET IN INFANTS WITH SEVERE INTRACTABLE DIARRHEA. *(E.)* Sherman, J. O.; Hamly, C.; and Khachadurian, A. K. (Child. Mem. Hosp., Chicago, Ill.). *J. Pediatr.* 86(4):518-523, 1975.

The efficacy of an oral elemental diet, Vivonex, in the treatment of severe intractable diarrhea in 27 infants (1 day to 9 months of age) and the diet's effect on nitrogen balance, plasma amino acids, lipid metabolism and acid-base balance were studied. The oral elemental diet (an 18% solution) consisted of crystalline amino acids, glucose, electrolytes, and vitamins and was administered by continuous nasogastric drip. The etiology of the diarrhea was nonspecific in 23 cases and bacterial in four. Diarrhea was controlled in 24 of 27 patients on the elemental diet, and the mean weight gain was 28 g/day. The mean length of treatment was 24 days. Some of the infants received standard Vivonex and others received Vivonex high-nitrogen diet. Those fed the standard diet received the recommended level of amino acids, but their nitrogen retention and weight gain were less than optimum. Feeding the high-nitrogen for-

mula (4.58 g/kg/day of amino acids) resulted in acceptable nitrogen retention and weight gain. The levels of individual plasma amino acids did not directly reflect the levels in the diet. Plasma triglyceride values remained within normal limits. SGPT was elevated in two patients after 13 days on the high-nitrogen diet and this may have indicated some transient liver toxicity. The higher levels of blood urea nitrogen in those receiving the high nitrogen diet probably reflected an excessive nitrogen intake. Although the oral elemental diet offers several advantages over total parenteral nutrition, the results suggest that an elemental diet specifically designed for use in infants should be developed. However, for patients with severe intractable diarrhea not responsive to conventional therapy, the presently available elemental diets represent a reasonable therapeutic alternative.

5019 AN APPLICATION OF SPECIAL ANALYSIS TO THE PROCESSING OF BIOELECTRIC VISCERAL SIGNALS. *(Fr.)* Tonkovic, S.; Penaud, J.; Thouvenot, J.; Mountafian, J. P. (Lab. Biomed. Electron., Fac. Electrotech., Zagreb, Yugoslavia). *Med. Biol. Eng.* 13(2):266-271, 1975.

Cutaneous abdominal electrodes were used to study overall bioelectric digestive activity in eight subjects, aged 20-25 yr. Two pairs of points symmetrically located on each side of the midline, one pair just below the rib cage and the other just above the pubis, were used for bipolar derivation of four signals. The sequential organization of the digestive motor process permits consideration of the signal as stationary within a defined interval. Therefore, the methods of spectral analysis may be applied to the values determined during the sequences of digestive activity. The power spectra produced by the eight subjects were statistically alike. A very good correlation was found between the frequencies recorded by this method and those reported in the literature and derived by various invasive physiological methods. The study of the distribution of energy contained in each derivation in a family of spectra permits determination of optimal electrode placement.

5020 CHRONIC DIARRHEA IN CHILDHOOD. A NEW LOOK AT AN OLD PROBLEM. *(E.)* Gall, D. G.; Hamilton, J. R. (Dep. Pediatr., Univ. Toronto, Canada). *Pediatr. Clin. North Am.* 21(4):1001-1017, 1974.

5021 CONGENITAL GRANULAR-CELL MYOBLASTOMA. *(E.)* Cussen, L. J.; MacMahon, R. A. (Dep. Pediatr., Monash Univ., Melbourne, Australia). *J. Pediatr. Surg.* 10(2):249-253, 1975.

5022 ANGIOGRAPHIC EVALUATION OF PATIENTS WITH CHRONIC GASTROINTESTINAL BLEEDING. *(E.)* Sheedy, II, P. F.; Fulton, R. E.; Atwell, D. T. (Mayo Clin., Rochester, Minn.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):338-347, 1975.

5023 CLINICAL PROBLEMS RELATED TO THE USE OF DRUGS IN MALNUTRITION. *(E.)* Poskitt, E. M. E. (Child. Hosp., Birmingham, England). *Proc. Nutr. Soc.* 33(3):203-207, 1974.

5024 GASTRIN IN GASTROINTESTINAL DISEASE. *(Fr.)* Brassinne, A. (Fac. Med., Univ. Liege, Belgium). *Rev. Med. Liege* 29(24):745-750, 1974.

5025 EFFECT OF METIAMIDE (INHIBITOR OF H_2 RECEPTORS FOR HISTAMINE) DURING ZOLLINGER-ELLISON SYNDROME. *(Fr.)* Bonfils, S.; Corbic, M.; Mignon, M.; Accary, J. P.; Bernades, P. (Bichat Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4): 292, 1974.

5026 SECONDARY HYPEROXALURIA AND INTESTINAL OXALIC ACID ABSORPTION IN GASTROINTESTINAL DISEASE. *(Ger.)* Caspary, W. F.; Tonissen, J.; Lankisch, P. G.; Schmidt, G. (Med. Clin., Univ. Gottingen, Germany). *Z. Gastroenterol.* 12(Suppl.): 425, 1974.

5027 IN SITU EVALUATION OF PROTEOLYSIS IN THE HUMAN STOMACH AND DUODENUM. *(Rus.)* Gorshkov, V. A. (Leningrad Inst. Sanit. Hyg., USSR). *Lab. Delo* (3):131-135, 1975

5028 EMBRYONIC ANTIGENS AND GASTROINTESTINAL TUMORS: CLINICAL SIGNIFICANCE. *(Rum.)* Belascu, M. (Reg. Hosp., Bistritsa, Rumania). *Cluj. Med.* 47(2):435-442, 1974.

5029 THE USE OF RADIOACTIVE ISOTOPES [^{59}Fe AND ^{51}Cr] TO STUDY THE RATE OF ERYTHROPOIESIS, ERYTHROCYTE SURVIVAL, AND OCCULT GASTROINTESTINAL BLEEDING IN PATIENTS WITH CHRONIC LYMPHOID LEUKEMIA. *(Rus.)* Sakhibov, Ia. D. (Cent. Clin. Hosp. No. 2, Minist. Railw., Moscow, USSR). *Probl. Gematol. Pereliv. Krovi* 20(2):30-35, 1975.

5030 EPIDEMIOLOGY OF Salmonella paratyphi B IN HUNGARY. *(Hung.)* Madar, J.; Adamis, E.; Milch, H.; Szentgali, J.; Borian, A.; Svidro, A.; Redey, B.; Gacs, M.; Olah, B. (Pest Cty. Public Health Epidemiol. Sta., Budapest, Hungary). *Egeszsegtudomany* 19(1):1-15, 1975.

5031 DIARRHEA IN INFANTS AND CHILDREN. *(Fr.)* deBrunhoff, M. (no affil.). *Pediatre (Clichy)* 10(48):277-290, 1974.

5032 THE USE OF INTERFERON TO PREVENT ACUTE RESPIRATORY DISEASES IN PATIENTS WITH INTESTINAL INFECTIONS. *(Rus.)* Feklisova, L. F.; Kuznetsov, V. P.; Vermgo, N. H.; Gureva, T. A. (M. F. Vladimirskii Res. Inst. Clin. Med., Moscow, USSR). *Vopr. Olch. Materin. Det.* 19(11):86, 1974.

5033 THE CLINICAL FEATURES OF ACUTE INFECTIOUS ENTEROCOLITIS IN ADULTS AND CHILDREN. *(Rus.)* Zhuravleva, L. V.; Maltseva, I. V. (State Inst. Postgrad. Med., Leningrad, USSR). *Sov. Med.* (1):154-155, 1975.

5034 FIBRINOGEN DEFICIENCY DURING THE COURSE OF TOXIC DIARRHEA. *(Pol.)* Kosinski, J.; Czarnecki, J. (W. Szenajch Munic. Child. Hosp., Torun, Poland). *Pediatr. Pol.* (2):239-240, 1975.

5035 GASTROINTESTINAL BLEEDING FROM A SURGICAL POINT OF VIEW. *(Ger.)* Heymann, H. (Ostadt Hosp., Hannover, Germany). *Med. Welt.* 25(36):1399-1404, 1974.

5036 GASTROINTESTINAL BLEEDING. *(Ger.)* Phlippen, R. (Med. Clin., Univ. Cologne, Germany). *Med. Welt.* 25(36):1394-1398, 1974.

5037 CHOLERA: REVIEW OF ITS ETIOLOGY, CLINICAL FEATURES, AND THERAPY. *(It.)* Rastelli, M. (Umberto I, Polyclin., Univ. Rome, Italy). *Clin. Eur.* 13(3):156-160, 1974.

5038 PREVENTION OF CHOLERA WITH DRUGS. *(It.)* Gaffi, A.; Businco, E.; De Pau, D. (Coll. Biol., Eur., Rome, Italy). *Clin. Eur.* 13(2):113-114, 1974.

5039 THE CLINICAL SIGNIFICANCE OF GASTROINTESTINAL HORMONES. *(Ger.)* Creutzfeldt, W. (Med. Clin., Univ. Gottinger, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:330-338, 1974.

5040 SOME PRINCIPLES FOR INTENSIVE TREATMENT OF CHOLERA AND CHOLERA-LIKE FORMS OF ACUTE INTESTINAL INFECTIONS. *(Rus.)* Bunin, K. V.; Sorinson, S. H. (I.M Sechenov Med. Inst., Moscow, USSR). *Klin. Med. (Mosk.)* 53(3):6-12, 1975.

5041 HYPEROXALURIA IN DISEASES OF THE GASTROINTESTINAL TRACT AND LIVER. *(Ger.)* Ruge, W.; Kohler, J.; Fromm, H.; Schindler, D.; Canzler, H. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:533-535, 1974.

5042 CHOLEPERITONEUM AND BILE PERITONITIS. *(Sp.)* Vidal Sans, J.; Avila, F.; Gomez Conde, S.; Zanon, V.; Olsina, J. (Francisco Franco, Munic. Hosp. Barcelona, Spain). *Rev. Esp. Enferm. Apar. Dig.* 45(3):295-306, 1975.

5043 CRONKHITE-CANADA SYNDROME. *(Ger.)* Bartelheimer, W.; Lux, G.; Ottenjann, R.; Elster, K. (Neuperlach Munic. Hosp., Munich, Germany). *Z. Gastroenterol.* 12(8):609-610, 1974.

5044 THREE CASES OF SEVERE TYPHOID FEVER OBSERVED DURING THE TYPHOID FEVER EPIDEMIC IN KRASNIK FABRYCZNY. *(Pol.)* Mardarowicz, C.; Szyxzko, B.; Patorska-Mach, E.; Modrzewska, R. (Acad. Med., Lublin, Poland). *Przegl. Epidemiol.* 28(2):227-232, 1974.

5045 SECONDARY INTOLERANCE OF DISACCHARIDES DURING INFANTILE DIARRHEA. *(Pol.)* Maszewska-Kuzniarz, K.; Karczewska, K.; Dyduch, A. (Acad. Med.; Zabrze, Poland). *Pediatr. Pol.* 49(12):1479-1484, 1974.

5046 EPIDEMIOLOGIC ASPECTS OF TYPHOID FEVER IN A COUNTRY WITH A LOW INCIDENCE RATE. II. INCIDENCE AND ASYMPTOMATIC INFECTIONS: EFFECTIVENESS OF DETECTION OF ASYMPTOMATIC INFECTIONS. *(Pol.)* Pietkiewicz, K.; Buczowski, Z. (Nav. Med. Inst., Gdansk, Poland). *Przegl. Epidemiol.* 28(3):333-338, 1974.

5047 TREATMENT OF DIGESTION AND ABSORPTION DISTURBANCES IN CHILDREN WITH CHRONIC MALNUTRITION. *(Pol.)* Planeta-Malecka, I.; Kluska, J. (Acad. Med., Lodz, Poland). *Wiad. Lek.* 28(4):275-284, 1975.

5048 PRIMARY LYMPHORETICULAR NEOPLASMS OF DIGESTIVE TRACT. *(Pol.)* Sulocka, J.; Kobierska, A.; Krajewska, B. (Acad. Med., Gdansk, Poland). *Wiad. Lek.* 28(4):265-268, 1975.

5049 DISACCHARIDASE ACTIVITY IN SMALL INTESTINAL MUCOSA DURING INFANTILE ENTEROPATHY. *(Pol.)* Hager-Malecka, B.; Karczewska, K.; Dyduch, A. (no affil.). *Pediatr. Pol.* 49(12):1471-1477, 1974.

5050 LACTIC DEHYDROGENASE ACTIVITY AND PYRUVIC ACID LEVEL IN BLOOD IN ACUTE DIARRHEA IN INFANTS. *(Pol.)* Paczos-Chadyma, E. (Inst. Pediatr., Lublin, Poland). *Pediatr. Pol.* 49(6):669-678, 1974.

5051 HISTOCHEMISTRY OF HUMAN DUODENAL MUCOUS MEMBRANE. *(Cz.)* Kocianova, J.; Lojda, Z. (Bulovka Hosp., Prague, Czechoslovakia). *Vnitr. Lek.* 20(7):652-657, 1974.

5052 USE OF INTESTINAL PROBES FOR PREVENTION AND TREATMENT OF POSTOPERATIVE ABDOMINAL COMPLICATIONS. *(Rus.)* Shcherbakov, A. A. (Res. Lab. Proctol., Moscow, USSR). *Khirurgiia (Mosk.)* (12): 91-96, 1974.

5053 MICROELEMENTS AND ELECTROLYTES IN THE SMALL INTESTINAL SECRETION OF PATIENTS WITH CHRONIC DIGESTIVE DISEASES. *(Rus.)* Revutsky, B. I.: Linevsky, Y. V. (Donetsk Med. Inst., USSR). *Vrach. Delo* 616(2):65-68, 1975.

5054 DECOMPRESSION GASTROSTOMY IN DIGESTIVE SURGERY: 100 CASES. *(Fr.)* Gignoux, M.; Bricard, H. (Surg. Clin. B, Univ. Caen, France). *Lyon Chir.* 71(1):58-60, 1975.

5055 THE ROLE OF THE LYMPH VESSEL SYSTEM IN DISEASES OF THE GASTROINTESTINAL TRACT. *(Ger.)* Meyer-Burg, J. (Steglitz Clin., Free Univ. Berlin, Germany). *Leber Magen Darm* 4(6):308-312, 1974.

5056 FUNCTIONAL ABDOMINAL COMPLAINTS: SYMPTOMS, BIOGRAPHY AND INTERRELATIONSHIPS. *(Ger.)* Schuffel, W. (Cent. Intern. Med. Pediatr., Univ. Ulm, Germany). *Leber Magen Darm* 4(3):66-103, 1974.

5057 PSYCHOSOMATIC PROBLEMS IN GASTROENTEROLOGY. *(Ger.)* Freyberger, H. (Univ. Hosp. Eppendorf, Hamburg, Germany). *Leber Magen Darm* 4(3):85-92, 1974.

5058 EFFECT OF CHEMOTHERAPY IN ACUTE SALMONELLOSIS ON THE DURATION OF FECAL SALMONELLA EXCRETION. A CATAMNESTIC STUDY IN THE CANTON OF ZUG (1969-1972). *(Ger.)* Hess, C. (Burgersp., Zug, Switzerland). *Schweiz. Rundschau Med.* 63(48):1430-1436, 1974.

5059 FUNCTIONAL RESULTS AFTER DUODENOPANCREATECTOMY (WITH SPECIAL CONSIDERATION OF IRON ABSORPTION, BREATH TEST, AND SERUM GASTRIN). *(Ger.)* Maurer, W.; Kaufmann, M.; Kaess, H.; Voirol, M.; Borel, G. A.; Deperrex, U.; Miller, G. (Burgersp., Solothurn, Germany). *Dtsch. Med. Wochenschr.* 99-(38):1863-1866, 1974.

5060 MEDICAL TREATMENT OF DIGESTIVE HEMORRHAGES. *(Sp.)* Kaufmann, P. (Fac. Med., Montevideo, Uruguay). *Cir. Urug.* 44(4):295-298, 1974.

5061 IATROGENIC DIGESTIVE HEMORRHAGES. *(Sp.)* Negrotto, J.; Niski de Rydel, R. (Fac. Med., Montevideo, Uruguay). *Cir. Urug.* 44(4):289-294, 1974.

5062 DIGESTIVE HEMORRHAGES: SURGICAL TREATMENT. *(Sp.)* Praderi, L. A. (Fac. Med., Montevideo, Uruguay). *Cir. Urug.* 44(4):299-305, 1974.

5063 SALIVARY GLAND CYLINDROMAS (ADENOID CYSTIC CARCINOMA). *(Fr.)* Baki, A.; Vaillant, J. M.; Brocheriou, C.; Laudenbach, P. (Salpetriere Hosp., Paris, France). *Bull. Cancer (Paris)* 61(4):501-508, 1974.

5064 TWO CASES OF TORSION OF THE GREATER OMENTUM. *(It.)* Balestrazzi, A. (Inst. Gen. Clin. Surg. Surg. Ther., Univ. Bari, Italy). *Chir. Gastroenterol.* 8(3):318-324, 1974.

5065 BIZARRE TUMORS (LEIOMYOBLASTOMA) OF DIGESTIVE TRACT SMOOTH MUSCULATURE. *(Sp.)* Diaz Watson, C.; Papamio Ruibal, A. (Cmdte. Manuel Fajardo Teaching Hosp., Havana, Cuba). *Rev. Cub. Cir.* 13(6):655-666, 1974.

5066 POLYPS IN THE UPPER GASTROINTESTINAL TRACT. *(Ger.)* Oltmanns, D.; Krautheim, J. (Acad. Med., Lubeck, Poland). *Med. Klin.* 69(43):1749-1755, 1974.

5067 INTESTINAL ABSORPTION OF VITAMIN D_3 AND CONVERSION INTO 25-OH D_3 DURING CHRONIC RENAL FAILURE. *(Fr.)* Bayard, F.; Bec, P.; Ton That, H.; Louvet, J. P.; Suc, J. M.; Boulard, C. (Fac. Med., Toulouse, France). *Rev. Med. Toulouse* 10(7):553-556, 1974.

5068 SURGERY AND PATHOLOGY OF THE BILE AND PANCREATIC DUCTS. *(Fr.)* Stalport, J. (no affil.). *Acta Gastroenterol. Belg.* 37(4/5):181-191, 1974.

5069 CLINICAL ASPECTS AND DIAGNOSIS OF OBSTRUCTIVE JAUNDICE IN TUMOR GROWTH. *(Rus.)* Shubin, B. M.; Rutoryan, E. S. (P. A. Gertsen Res. Inst. Oncol., Moscow, USSR). *Vopr. Onkol.* 21(2):40-44, 1975.

5070 THE CONGENITAL NATURE OF THE STENOSIS DUE TO THE DIAPHRAGMATIC COMPRESSION OF THE CELIAC ARTERY. A NEW ARGUMENT SUPPORTING THIS INTERPRETATION: THE CASES OF TWO SISTERS. *(Fr.)* Warter, J.; Storck, D.; Gillet, B.; Tongio. J. (Univ. Hosp. Cent., Strasbourg, France). *J. Med. Strasbourg* 5(5):369-375, 1974.

5071 INTESTINAL FORM OF CYTOMEGALY IN YOUNG CHILDREN. *(Rus.)* Pianov, R. P. (Gatchinsk Cent. Reg. Hosp., Leningrad Reg., USSR). *Vopr. Okhr. Materin. Det.* 18(12):25-29, 1974.

5072 SALMONELLA CARRIER STATE IN CHILDREN. *(Rus.)* Greshilo, M. S.; Silina, E. D. (N. I. Pirogov Med. Inst., Vinnitsa, USSR). *Vopr. Okhr. Materin, Det.* 18(12):21-24, 1974.

5073 DETERMINATION OF THE PHYSICOCHEMICAL NATURE OF SPECIFIC ANTIBODIES IN VARIOUS FORMS AND COURSES OF DYSENTERY IN CHILDREN. *(Rus.)* Nesterina, L. F.; Vasilieva, I. G. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Vopr. Okhr. Materin. Det.* 18(12):16-20, 1973.

5074 STUDIES ON CHOLERA CRISIS-INHIBITING FACTORS: (2). PURIFICATION AND CHARACTERIZATION OF ACTIVE SUBSTANCES FROM SMALL INTESTINES OF IMMUNIZED RABBITS. *(Jpn.)* Morimoto, T.; Yamamoto, M.; Maruyama, S.; (Teikoka Hormone Mfg. Co. Ltd., Kawasaki, Japan). *J. Pharm. Soc. Jpn.* 94(12):1560-1565, 1974.

5075 PENETRATION OF Shigella INTO THE EPITHELEAL CELLS AS AN IMPORTANT CONDITION FOR THE DEVELOPMENT OF AN INFECTIOUS PROCESS. *(Rus.)* Petrovskaia, V. G. (Gamaleia Inst. Epidemiol. Microbiol., Moscow, USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* 616(9):24-28, 1974.

5076 STUDY OF THE ETIOLOGICAL ROLE OF Klebsiella IN ACUTE INTESTINAL DISEASES. *(Rus.)* Samokhina, Z. F.; Gurevich, G. T.; Kuzina, M. F. (Minsk. Med. Inst., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* 616(8):64-67, 1974.

5077 LORAZEPAM IN THE MANAGEMENT OF GASTROINTESTINAL NEUROSES. *(E.)* El Roubi, A. (Dep. Med., Cairo Univ., Egypt). *J. Int. Med. Res.* 3(2):134-137, 1975.

5078 MARASMUS AND Pneumocystis carinii PNEUMONIA IN INSTITUTIONALISED INFANTS. OBSERVATIONS DURING AN ENDEMIC. *(E.)* Dutz, W.; Jennings-Khodadad, E.; Post, C.; Kohour, E.; Nazarian, I.; Esmaili, H. (Dep. Pathol., Univ. Pahlave, Shiraz, Iran). *Z. Kinderheilkd.* 117:241-258, 1974.

5079 THIORIDAZINE-INDUCED DIARRHOEA. *(E.)* Mitchell, A. B. S. (Charing Cross Hosp., London, England). *Postgrad. Med. J.* 51(593):182-183, 1975.

5080 NECROTIZING ENTERCOLITIS. *(E.)* Calahane, S. F.; Moore, B. (Child. Hosp., Dublin, Ireland). *Ir. J. Med. Sci.* 144(3):85-91, 1975.

5081 OCCULT GASTROINTESTINAL BLEEDING. AN EVALUATION OF AVAILABLE DIAGNOSTIC METHODS. *(E.)* Richardson, J. D.; McInnis, W. D.; Ramos, R.; Aust, J. B. (Univ. Texas Health Sci. Cent., San Antonio). *Arch. Surg.* 110(5):661-665, 1975.

5082 ENTEROPATHOGENIC Esch. coli GASTROENTERITIS IN PREMATURE INFANTS AND CHILDREN TREATED WITH FOSFOMYCIN. *(E.)* Baquero, F.; Canedo, T.; Rodriguez, A.; Jaso, E. (La Paz Pediatr. Clin., Auton. Univ. Madrid, Spain). *Arch. Dis. Child.* 50(5):367-372, 1975.

5083 REVERSIBLE FINGER CLUBBING IN A CASE OF PURGATIVE ABUSE. *(E.)* Silk, D. B. A.; Gibson, J. A.; Murray, C. R. H. (St. Bartholomew's Hosp., London, England). *Gastroenterology* 68(4):790-794, 1975.

5084 NEONATAL NECROTIZING ENTEROCOLITIS. EVOLUTION OF NEW PRINCIPLES IN MANAGEMENT. *(E.)* Wayne, E. R.; Burrington, J. D.; Hutter, J. (Child. Hosp., Denver, Colo.). *Arch Surg.* 110(5):476-480, 1975.

5085 CURRENT MANAGEMENT OF UPPER GASTROINTESTINAL BLEEDING. *(E.)* Yajko, R. D.; Norton, L. W.; Eiseman, B. (Denver Gen. Hosp., Colo.). *Ann. Surg.* 181(4):474-480, 1975.

5086 SURGICAL TREATMENT OF TYPHOID PERFORATION. *(E.)* Welch, T. P.; Martin, N. C. (Dep. Surg., Univ. Chiang Mai, Thailand). *Lancet* 1(7915):1078-1080, 1975.

5087 BIOCHEMICAL CHANGES IN SERUM FREE AMINOACIDS AND SERUM TRANSAMINASES IN INFANTILE GASTRO-ENTERITIS. *(E.)* Al-Nagdy, S. A.; Refai, F. M.; Khattab, A. K. (Dep. Biochem., Ein-Shams Univ, Cairo, Egypt). *Int. J. Biochem.* 5(3):251-256, 1974.

5088 THE USE OF A CARBOHYDRATE-FREE FEEDING FORMULA IN AUSTRALIAN ABORIGINAL CHILDREN WITH CHRONIC DIARRHOEA. *(E.)* Gracey, M.; Henderson, R. (Princess Margaret Child. Med. Res. Found., Perth, Australia). *Aust. Paediatr. J.* 10(3):164-167, 1974.

5089 DIETARY FIBRE AND 'PRESSURE DISEASES'. *(E.)* Burkitt, D. P. (Med. Res. Counc., London, England). *J. R. Coll. Physicians Lond.* 9(2):138-147, 1975.

5090 INTESTINAL DISACCHARIDASES IN HYPOPROTEINAEMIC STATES. *(E.)* Rastogi, M.; Misra, R. C.; Sarin, G. S.; Srivastava, P. N.; Chuttani, H. K. (Maulana Azad Med. Coll., New Delhi, India). *J. Assoc. Physicians India* 22(12):889-892, 1974.

5091 EFFECTS OF FRUCTOSE ON FERRIC AND FERROUS IRON ABSORPTION IN MAN. WITHIN-SUBJECT STUDY WITH THE ^{59}Fe-ABSORPTION WHOLE-BODY-RETENTION TEST IN SUBJECTS WITH NORMAL AND DEPLETED IRON STORES. *(E.)* Heinrich, H. C.; Gabbe, E. E.; Bruggemann, J.; Oppitz, K. H. (Univ. Hosp. Eppendorf, Hamburg, Germany). *Nutr. Metab.* 17(4):236-248, 1974.

5092 THE RELATIONSHIP OF THE FECES PROTEIN PARTICLES TO RICE PROTEIN BODIES. *(E.)* Tanaka, Y.; Hayashida, S.; Hongo, M. (Dep. Agric. Chem., Kyushu Univ., Fukuoka, Japan). *Agric. Biol. Chem.* 39(2):575-578, 1975.

5093 NATURAL HISTORY AND EXPERIENCE WITH DIAGNOSIS AND TREATMENT OF THE ZOLLINGER-ELLISON SYNDROME. *(E.)* Thompson, J. C.; Reeder, D. D.; Villar, H. V.; Fender, H. R. (Univ. Texas Med. Branch, Galveston). *Surg. Gynecol. Obstet.* 140(5):721-739, 1975.

5094 ALCOHOLISM AND THE RISK OF CANCER. *(E.)* Lowenfels, A. B. (New York Med. Coll., Valhalla). *Ann. NY Acad. Sci.* 252:366-373, 1975.

5095 MODEL FOR THE GROWTH OF A SOLID IN VITRO TUMOR. *(E.)* Deakin, A. S. (Dep. Applied Math., Univ. West. Ontario, Canada). *Growth* 39(1): 159-165, 1975.

5096 TOTAL HUMAN BODY PROTEIN SYNTHESIS IN RELATION TO PROTEIN REQUIREMENTS AT VARIOUS AGES. *(E.)* Young, V. R.; Steffee, W. P.; Pencharz, P. B.; Winterer, J. C.; Scrimshaw, N. S. (Dep. Nutr. Food Sci., Massachusetts Inst. Tech., Cambridge). *Nature* 253(5488):192-193, 1975.

5097 SULFATE-INDUCED DIARRHEA. *(Ger.)* Schmidt-Kessen, W.; Witte, O. (Inst. Balneol. Clim. Physiol., Univ. Freiburg/Breisgau, Germany). *Z. Gastroenterol.* 13(1):12-18, 1975.

5098 DEFICIENCY OF DISACCHARIDASES AND ALKALINE PHOSPHATASE IN SMALL INTESTINE MUCOSA IN PATIENTS WITH SOME ACUTE INTESTINAL INFECTIONS. *(Rus.)* Bobkova, S. D.; Shalygina, P. B.; Iushchuk, N. D. (Cent. Res. Inst. Epidemiol., Moscow, USSR). *Vopr. Med. khim.* 20(3):243-248, 1974.

5099 SECRETORY AND MORPHOLOGICAL DISORDERS OF THE STOMACH AND DUODENUM IN PATIENTS WITH CHRONIC HEPATITIS AND CIRRHOSIS OF THE LIVER. *(Rus.)* Iakhontova, O. I.; Krutskikh, E. V.; Valenkevich, L. N. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Klin. Med. (Mosk.)* 52(7):67-72, 1974.

5100 QUANTITATIVE FUNCTIONAL EXPLORATION OF THE SALIVARY GLANDS. *(Fr.)* Ingrand, J.; Danchot, J.; Menkes, C. J.; Delbarre, F.; Roucayrol, J. C. (Cochin Hosp., Paris, France). *Nouv. Presse. Med.* 4(6):395-400, 1975.

5101 ACINIC CELL ADENOCARCINOMA OF THE MINOR SALIVARY GLANDS: A LIGHT MICROSCOPIC AND ULTRASTRUCTURAL STUDY OF A CASE. *(Jpn.)* Esaki, T.; Katoch, Y.; Yoshida, M.; Takeuchi, J. (Dep. Pathol., Aichi-Gakuin Univ., Nagoya, Japan). *Jpn. J. Cancer Clin.* 20(7):553-560, 1974.

5102 FUNCTIONAL MORPHOLOGY OF THE SMALL INTESTINE MUCOSA IN PATIENTS WITH CHOLERA. (FROM ASPIRATION BIOPSY FINDINGS). *(Rus.)* Shalygina, N. B. (Cent. Res. Inst. Epidemiol., Moscow, USSR). *Arkh. Patol.* 36(11):16-22, 1974.

5103 FINDINGS ON ASPIRATION BIOPSIES FROM THE INTESTINAL MUCOSA IN PHENYLKETONURIA. *(Ger.)* Cobet, G.; David, H.; Mucke, D.; Nitz, I.; Robbe, K. (Dep. Med., Humboldt Univ., Berlin, Germany). *Paediatr. Grenzgeb.* 13(1):13-22, 1974.

5104 GASTROINTESTINAL PERFORATIONS IN INFANCY. ANATOMIC AND ETIOLOGIC GAMUTS. *(E.)* Tucker, A. S.; Soine, L.; Izant, Jr., R. J. (Univ. Hosp., Cleveland, Ohio). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4):755-763, 1975.

5105 TRANSMISSIBLE ENTEROCOLITIS IN HAMSTERS CAUSED BY TYZZER'S ORGANISM. *(E.)* Nakayama, M.; Saegusa, J.; Itoh, K.; Kiuchi, Y.; Tamura, T.; Ueda, K.; Fujiwara, K. (Inst. Med. Sci., Univ. Tokyo, Japan). *Jpn. J. Exp. Med.* 45(1):33-41, 1975.

5106 ON THE BIOCHEMICAL BASIS OF HEREDITARY FRUCTOSE INTOLERANCE. *(E.)* Koster, J. F.; Slee, R. G.; Fernandes, J. (Med. Fac., Erasmus Univ., Rotterdam, The Netherlands). *Biochem. Biophys. Res. Commun.* 64(1):289-294, 1975.

5107 TYPHOID γG, γM AND γA ANTIBODIES IN PERSONS WITH DIFFERENT RELATION TO THE TYPHOID INFECTION. *(E.)* Ciznar, I.; Farbakyova, G.; Draskovicova, M.; Karolcek, J. (Res. Inst. Epidemiol. Microbiol., Bratislava, Czechoslovakia). *Zentralbl. Bakteriol. [Orig. A]* 231(1-3):108-115, 1975.

5108 LOWERED SERUM CHOLESTEROL FOLLOWING THE INGESTION OF A HYDROPHILIC COLLOID. *(E.)* Lieberthal, M. M.; Martens, R. A. (Bridgeport Hosp., Conn.). *Am. J. Dig. Dis.* 20(5):469-474, 1975.

5109 VASCULAR TUMORS OF THE SALIVARY GLANDS IN CHILDREN. *(E.)* Hebert, G.; Ouimet-Oliva, D.; Ladouceur, J. (Notre Dame Hosp., Montreal, Canada). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4):815-819, 1975.

5110 SURGICAL MANAGEMENT OF INFANTILE ULCERATIVE ENTERITIS. *(E.)* Miller, R. C.; (Univ. Mississippi Med. Cent., Jackson). *J. Pediatr. Surg.* 10(3):367-373, 1975.

5111 MANAGEMENT OF PAROTID GLAND TUMORS IN INFANTS AND CHILDREN. *(E.)* Chong, G. C.; Beahrs, O. H.; Chen, M. L. C.; Hayles, A. B. (Mayo Grad. Sch. Med., Univ. Minnesota, Rochester). *Mayo Clin. Proc.* 50(5):279-283, 1975.

5112 AN OUTBREAK OF GASTROENTERITIS DUE TO E. coli 142 IN A NEONATAL NURSERY. *(E.)* Boyer, K. M.; Petersen, N. J.; Farzaneh, I.; Pattison, C. P.; Hart, M. C.; Maynard, J. E. (U.S. Public Health Serv., Phoenix, Ariz.). *J. Pediatr.* 86(6): 919-927, 1975.

5113 TREATMENT OF ACRODERMATITIS ENTEROPATHICA WITH ZINC SULFATE. *(E.)* Robertson, A. F.; Sotos, J. (Med. Coll., Augusta, Ga.). *Pediatrics* 55(5):738-739, 1975.

5114 DIARRHOEA AND DIGESTIVE ENDOCRINE TUMOURS. *(E.)* Rambaud, J. C.; Matuchansky, C. (no affil.). *Clin. Gastroenterol.* 3(3):657-670, 1974.

5115 ZOLLINGER-ELLISON SYNDROME: NATURAL HISTORY AND DIAGNOSIS. *(E.)* Bonfils, S.; Bernades, P. (no affil.). *Clin. Gastroenterol.* 3(3): 539-557, 1974.

5116 MULTIPLE ENDOCRINE ADENOMATOSIS; MULTIPLE HORMONE-PRODUCING TUMOURS, A FAMILIAL SYNDROME. *(E.)* Wermer, P. (no affil.). *Clin. Gastroenterol.* 3(3):671-684, 1974.

5117 THE ENDOCRINE CELLS OF THE GI TRACT: ORIGENS, MORPHOLOGY AND FUNCTIONAL RELATIONSHIPS IN HEALTH AND DISEASE. *(E.)* Pearse, A. G. E. (no affil.). *Clin. Gastroenterol.* 3(3):491-510, 1974.

5118 PATHOLOGY OF ENDOCRINE TUMOURS OF THE GI TRACT. *(E.)* Martin, E. D.; Potet, F. (no affil.). *Clin. Gastroenterol.* 3(3):511-532, 1974.

5119 FATAL Yersinia enterocolitica SEPTICAEMIA. *(E.)* Josefsson, K.; Linberg, A. (Cent. Hosp., Halmstad, Sweden). *Scand. J. Infect. Dis.* 7(1):76-77, 1975.

5120 NATURAL HISTORY AND DIAGNOSIS OF THE CARCINOID SYNDROME. *(E.)* Grahame-Smith, D. G. (no affil.). *Clin. Gastroenterol.* 3(3):575-594, 1974.

5121 NON-B ISLET TUMOURS AND THE SYNDROME OF WATERY DIARRHOEA, HYPOKALAEMIA AND HYPOCHLORHYDRIA. *(E.)* Verner, J. V.; Morrison, A. B. (no affil.). *Clin. Gastroenterol.* 3(3):595-608, 1974.

5122 MODERN CONCEPTS IN THE MANAGEMENT OF GASTROJEJUNOCOLIC FISTULA. *(E.)* Hodgson, J.; Medina, O. F.; Teodorescu, M.; Messe, A. A. (Bronx VA Hosp., N.Y.). *Mt. Sinai J. Med. NY* 42(3): 239-244, 1975.

5123 HEREDITARY TELANGIECTASIA MANIFESTED AS GASTROINTESTINAL BLEEDING WITHOUT EXTERNAL VISIBLE TELANGIECTASIA. *(E.)* McCaffery, Jr., T. D.; Lilly, J. O. (Louisiana State Univ. Med. Cent., New Orleans). *Am. J. Gastroenterol.* 63(4): 327-332, 1975.

5124 SALIVARY GLAND DISEASE. *(E.)* Chisholm, D. M.; Mason, D. K. (Dep. Oral Med. Pathol., Univ. Glasgow, Scotland). *Br. Med. Bull.* 31(2):156-158, 1975.

5125 RECENT ADVANCES IN PHYSIOLOGY OF SALIVARY GLANDS. *(E.)* Garrett, J. R. (King's Coll. Hosp. Med. Sch., London, England). *Br. Med. Bull.* 31(2):152-155, 1975.

5126 THE INVESTIGATION OF ACUTE UPPER GASTROINTESTINAL HAEMORRHAGE. *(E.)* Forrest, J. A. H.; Finlayson, N. D. C. (R. Infirm., Edinburgh, Scotland). *Br. J. Hosp. Med.* 12(2):160-167, 1974.

5127 SEPTICAEMIA DUE TO Yersinia enterocolitica IN A NON-COMPROMISED HOST. *(E.)* Eriksson, M.; Olcen, P. (Reg. Hosp., Orebro, Sweden). *Scand. J. Infect. Dis.* 7(1):78-80, 1975.

5128 INCREASED COPPER/ZINC RATIOS IN ACRODERMATITIS ENTEROPATHICA. *(E.)* Strain, W. H.; Hirsh, F. S.; Michel, B. (Cleveland Metrop. Gen. Hosp., Ohio). *Lancet* 1(7917):1196-1197, 1975.

5129 ZINC DEFICIENCY AND CELLULAR IMMUNE DEFICIENCY IN ACRODERMATITIS ENTEROPATHICA. *(E.)* Endre, L.; Katona, Z.; Gyurkovits, K. (Univ. Med. Sch., Szeged, Hungary). *Lancet* 1(7917):1196, 1975.

5130 EMERGENCY ELECTROCOAGULATION FOR UPPER GI BLEEDING. *(E.)* Agrawal, R. M.; Cincala, R. P.; Brodmerkel, Jr., G. J. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Gastrointest. Endosc.* 21(4):183, 1975.

See also, 3799, 4201, 4202, 4203, 4207, 4208, 4210, 4297, 4311, 4317, 4347, 4350, 4553, 4608.

GENERAL

Parasitology

5131 AMEBIC COLITIS MISTAKEN FOR INFLAMMATORY BOWEL DISEASE. *(E.)* Tucker, P. C.; Webster, P. D.; Kilpatrick, Z. M. (Med. Coll. Georgia, Augusta). *Arch. Intern. Med.* 135(5):681-685, 1975.

Ten cases of patients with amebic colitis which had been mistaken for inflammatory bowel disease was described. Similarities in clinical findings made diagnosis difficult; multiple stool examinations failed to demonstrate ova or trophozoites of *Entamoaba histolytica*. The indirect hemagglutination test was found to be a reliable diagnostic test in the evaluation of patients with inflammatory bowel disease. Substances taken early for symptomatic relief of diarrhea may interfere with the detection of amebas and lead to false-negative results; thus, amebiasis should be considered during the initial evaluation of patients with chronic diarrhea, especially if the diarrhea is bloody. The routine use of the serological test for amebiasis is recommended, followed by amebiasis treatment for all patients with colitis who have diagnostic indirect hemagglutination titers.

5132 BILHARIZIASIS: SIX CASES. *(Fr.)* St.-Yves, C.; Tremblay, M.; Garon, G.; Rousseau, B. (Hosp. Cent., Univ. Laval, Quebec, Canada). *Vie Med. Can. Fr.* 3(1):47-53, 1974.

Cases of bilharziasis caused by *Schistosoma mansoni* are reported. All six patients (five men, one woman, aged 21-58 yr) had just returned from a stay in Chad of 2 to 18 yr. Symptoms included asthenia, anorexia, nausea, vomiting, hyperthermia, abdominal cramps, diarrhea, bloody stool, icterus, and weight loss; two patients showed no symptoms at all. Examination of the urine showed no evidence of parasites in any of the cases, and examination of the stool showed only one egg in only one case. Rectal mucosal biopsy in five patients gave positive results; histological examination revealed tuberculoid granulomas in the submucosa, the larger of which contained eggs or egg fragments. The patient who did not undergo rectal mucosa biopsy had previously had a liver needle biopsy which proved the diagnosis of *S. mansoni* infection in addition to viral hepatitis. In all cases, there was peripheral eosinophilia, ranging from 730 - 1558 cells/ml. All patients were treated with niridazole, which produced side effects of headaches, myalgia, anorexia, nausea, vomiting, slowing of intestinal transit, and minor electrocardiogram changes. The absence of major side effects on the central nervous system is attributed to the mitigating effects of diazepam during treatment and the absence of portal hypertension at the time of treatment. The side effects were consistently more severe for the two patients with liver involvement. Eosinophilia increased during treatment, but gradually returned to normal several months after treatment.

5133 TRENDS IN PARASITOLOGICAL RESEARCH. *(Ger.)* Piekarski, G. (Inst. Med. Parasitol., Univ. Bonn-Venusberg, Germany). *Z. Parasitenkd.* 45(2):91-108, 1974.

5134 HELMINTHS AND ZOONOSES. *(Ger.)* Petrovic, Z. (Fac. Vet. Med., Belgrade, Yugoslavia). *Z. Parasitenkd.* 45(2):221-225, 1974.

5135 INTESTINAL PARASITES IN RESIDENTS OF DRESDEN WHO HAVE RETURNED FROM THE TROPICS. *(Ger.)* Muller, W. A. (Inst. Med. Microbiol. Epidemiol., Dresden, Germany). *Z. Gesamte Inn. Med.* 30(7):269-271, 1975.

5136 SAMPLING THE INCIDENCE OF INTESTINAL HELMINTHS OF POPULATIONS IN VARIOUS CLIMATIC AND GEOGRAPHIC REGIONS. 1. THE ORGANISATION AND EFFECTIVENESS OF SAMPLING THE POPULATION FOR HELMINTHIC INFESTATIONS. *(Rus.)* Kurpakov, N. A. (E. I. Martsinovskii Inst. Med. Parasitol. Trop. Med., Moscow, USSR). *Med. Parazitol.* 43(3):265-269, 1974.

5137 AMEBIC ABSCESS OF THE LIVER AND SPLEEN IN PREGNANCY AND THE PUERPERIUM. *(E.)* Wagner, V. P.; Smale, L. E.; Lischke, J. H. (Kern County Gen. Hosp., Bakersfield, Calif.). *Obstet. Gynecol.* 45(5):562-565, 1975.

5138 A REVIEW OF ECHINOCOCCAL DISEASE. *(E.)* Lewis, Jr., J. W.; Koss, N.; Kerstein, M. D. (Yale Univ. Sch. Med., New Haven, Conn.). *Ann. Surg.* 181(4):390-396, 1975.

5139 UNUSUAL COMPLICATION OF LIVER ECHINOCOCCOSIS: OBSTRUCTION OF THE COMMON BILE DUCT WITH ECHINOCOCCAL CYSTS. *(Ser.)* Begic-Janev, A.; Jelenkovic, M.; Stajic, M.; Lastic-Maletic, S.; Korajkic, M. (Sch. Med., Univ. Belgrade, Yougoslavia). *Srp. Arhir.* 102(6):485-491, 1974.

5140 INTESTINAL PROTAZOAL INFECTION IN THE INHABITANTS OF DRESDEN. *(Ger.)* Muller, W. A. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Z. Gesamte. Inn. Med.* 30(5):209-214, 1975.

5141 INTESTINAL HELMINTH INFESTATION IN THE INHABITANTS OF DRESDEN. *(Ger.)* Muller, W. A. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Z. Gesamte. Inn. Med.* 30(6):239-247, 1975.

5142 DIAGNOSIS AND TREATMENT OF AMEBIC LIVER ABSCESS. *(Fr.)* Laverdant, C. (Begin Army Hosp., St. Mande, France). *Ann. Med. Interne (Paris)* 125(11):803-809, 1974.

5143 A NEW ANTHELMINTIC MEBENAZOLE. *(Fr.)* Bekhti, A. (Dep. Clin. Med. Pathol., Univ. Liege, Belgium). *Acta Gastroenterol. Belg.* 37(6): 302-306, 1974.

5144 HEPATOSPLENIC SCHISTOSOMIASIS: GENERAL PATHOGENESIS, BIOLOGICAL DIAGNOSIS, AND TREATMENT. *(Fr.)* Lapierre, J. (Cochin Hosp., Paris, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(4): 341-353, 1974.

5145 CLINICAL MANIFESTATIONS OF HEPATOSPLENIC SCHISTOSOMIASIS. *(Fr.)* Gentilini, M.; Garabiol, B. (Pitie-Salpetriere Univ. Hosp. Cent., Paris, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(4):331-339, 1974.

5146 CLINICAL PICTURE OF AMEBIC ABSCESS OF THE LIVER IN THE LIGHT OF OBSERVATIONS MADE AT GASTROENTEROLOGIC CENTERS IN TROPICAL COUNTRIES. *(Pol.)* Lachecki, Z. (G. Narutowicz Munic. Hosp., Krakow, Poland). *Wiad. Parazytol.* 20(4):499-503, 1974.

5147 TREATMENT OF AMEBIASIS WITH ORAL METRONIDAZOLE SOLUTIONS. *(Fr.)* Sankale, M.; Coly, D.; Niang, I. (Fac. Med. Pharm., Dakar, Senegal). *Therapie* 29(3):411-415, 1974.

5148 DIAGNOSIS AND TREATMENT OF COMPLICATED HYDATID DISEASE OF THE LIVER. *(Rus.)* Lagutin, V. D. (Munic. Hosp. No. 48, Moscow, USSR). *Khirurgiia (Mosk.)* 616(3):80-84, 1974.

5149 HEPATIC AMEBIASIS WITH DOMINANT PLEURAL AND PULMONARY SYMPTOMS. REPORT OF TWO CASES. *(Fr.)* Bekhti, A. (Dep. Clin. Med. Pathol., Univ. Liege, Belgium). *Acta Gastroenterol. Belg.* 37(4/5): 225-235, 1974.

5150 NICARBAZINE IN SCHISTOSOME INFECTIONS: I. ANTIBODY FORMATION IN MICE AND HAMSTERS INFECTED WITH Schistosoma mansoni. *(E.)* Hillyer, G. V.; Reyes, C. N.; Hernandez-Almenas, N. (Dep. Biol., Univ. Puerto Rico, Rio Pedras). *Exp. Parasitol.* 37(3):442-448, 1975.

5151 DOGS AND INTESTINAL PARASITES: A PUBLIC HEALTH PROBLEM. *(E.)* Seah, S. K. K.; Hucal, G.; Law, C. (Queen Mary Vet. Hosp., McGill Univ., Montreal, Canada). *Can. Med. Assoc. J.* 112(10):1191-1194, 1975.

5152 THE BIOCHEMISTRY OF HELMINTHS. *(E.)* von Brand, T. (no affil.). *Z. Parasitenkd.* 45(2):109-124, 1974.

5153 THE ECONOMICS OF HUMAN PARASITIC INFECTIONS. *(E.)* Olivier, L. J. (no affil.). *Z. Parasitenkd.* 45(2):197-210, 1974.

5154 AMEBIC PERICARDIAL EFFUSION: A RARE COMPLICATION OF AMEBIC LIVER ABSCESS. *(E.)* Agrawal, B. V.; Somani, P. N.; Khanna, M. N.; Srivastava, P. K.; Jha, B. N.; Verma, S. P. (Inst. Med. Sci., Banaras Hindu Univ., Varanasi, India). *Am. Surg.* 41(6):373-377, 1975.

5155 SURGICAL INTERVENTION IN INTESTINAL AMEBIASIS. *(E.)* Latimer, R. G. (Santa Barbara Med. Clin., Calif.). *Am. Surg.* 41(6):385-390, 1975.

5156 MASSIVE DEVELOPMENT OF AMEBAS IN THE LARGE INTESTINE. *(E.)* Lesh, F. A. (no affil.). *Am. J. Trop. Med. Hyg.* 24(3):383-392, 1975.

5157 Trichinella spiralis: MORPHOLOGICAL CHARACTERISTICS OF MALE AND FEMALE INTESTINE-INFECTING LARVAE. *(E.)* Kozek, W. J. (California Primate Res. Cent., Univ. California, Davis). *Exp. Parasitol.* 37(3):380-387, 1975.

5158 Fasciola hepatica: DEVELOPMENT OF CAECAL EPITHELIUM DURING MIGRATION IN THE MOUSE. *(E.)* Bennett, C. E. (Zool. Dep., Queen's Univ., Belfast, Northern Ireland). *Exp. Parasitol.* 37(3): 426-441, 1975.

See also, 4193, 4242, 4282.

GENERAL

Regional Enteritis

5159 IN VITRO STUDIES OF ULCERATIVE ILEOJEJUNITIS. *(E.)* Klaeveman, H. L.; Gebhard, R. L.; Sessoms, C.; Strober, W. (Natl. Inst. Arthritis Metab. Dig. Dis., Bethesda, Md.). *Gastroenterology* 68(3):572-582, 1975.

Behavior in organ culture and local immunoglobulin responses were studied in tissue from a patient with chronic ulcerative ileojejunitis. The patient was a 42-yr-old Jewish male who presented with diarrhea and abdominal distention, digital clubbing and trace pedal edema. Small bowel x-rays showed an abnormal loop in the left upper quadrant with nodular indentations. Peroral jejunal biopsy showed complete villous atrophy with cuboidal pyknotic epithelial cells and a dense infiltrate of plasma cells, eosino-

phils and histiocytes. Intestinal biopsies were taken with the instrument placed fluoroscopically at the ligament of Treitz, rinsed in Krebs-Ringer bicarbonated buffer, cut into pieces 1-2 cm in diameter, and cultured in the presence and absence of gluten peptides. Specimens were also incubated in L-^{14}C-leucine for 90 min, the tissue homogenized, and the resulting homogenate incubated for 3 hr with anti-IgA antibodies linked to bromacetyl cellulose. Leucine incorporation into IgA after a 7 day gluten-containing diet was 32,647 counts/min/mg protein in comparison to 5830 counts/min/mg protein in eight normal individuals. Studies while the patient was on a gluten-free diet still showed an elevated count (34,000) but steroid therapy brought the values doen to 6814 after two months. Initial and 48 hr alkaline phosphatase values for organ cultured tissues were 51 and 262 μM *p*-nitrophenyl phosphate formed/g tissue/min, resp., after the gluten free diet and 108 and 497, resp., after the gluten-containing diet. In culture studies performed when the patient was on a gluten free diet, before the institution of corticosteroid therapy, initial alkaline phosphatase increased from 51 - 262 U. A similar increase to 242 U occured when gluten was present in the medium. It is concluded that chronic ulcerative ileojejunitis is a complication of gluten-sensitive enteropathy related to a non-gluten dependent pathological process involving the local immunological system.

5160 THE THYROID IN ULCERATIVE COLITIS AND CROHN'S DISEASE. IV. THYROID HORMONE BINDING PROTEINS. *(E.)* Jarnerot, G.; Truelove, S. C.; von Schenck, H. (Radcliffe Infirm., Oxford, England). *Acta. Med. Scand.* 197(1/2):95-98, 1975.

The serum concentrations of the thyroid binding proteins were studied in patients with ulcerative colitis or Crohn's disease. Of the 20 patients studied, 11 had ulcerative colitis, of whom 8 had universal, and 3 distal involvement. Of the nine with Crohn's disease, 4 had colonic disease, three small bowel disease, and two ileocecal inflammation. Ten of the patients were on corticosteroid treatment (30-60 mg prednisolone/day). Controls were 20 healthy subjects not on any medication. Serum thyroxine binding globulin and thyroxine binding prealbumin were measured by agar gel electrophoresis and serum albumin was measured with bromcresol green. Serum thyroxine was determined by competitive protein binding, and thyroid stimulating hormone by radioimmunoassay. Thyroxine binding globulin levels were significantly higher in patients not taking corticosteroids than in controls (28.1 and 19.3 μg/100 ml, resp.), while thyroxine binding prealbumin levels were significantly lower (90.9 and 142.8 μg/100 ml, resp.). Thyroxine levels did not differ significantly, nor did triiodothyronine uptake. Patients receiving corticosteroids had significantly lower serum albumin levels than those not recieving them (32.3 and 40.3 μg/100 ml, resp.). The correlation between thyroxine binding prealbumin and albumin was significant when estimated for patients and controls as one group. Patients with no corticosteroid therapy had a significantly higher free thyroxine index than the controls (4.4 and 3.9 μg/100 ml, resp.). The thyroid stimulating hormone level was normal in all patients and controls. It is concluded that in ulcerative colitis and Crohn's disease changes can occur in serum concentrations of thyroxine binding proteins which may cause alterations in thyroid tests.

5161 THE THYROID IN ULCERATIVE COLITIS AND CROHN'S DISEASE. III. THE DAILY FRACTIONAL TURNOVER OF THYROXINE. *(E.)* Jarnerot, G.; Truelove, S. C.; Warner, G. T. (Radcliffe Infirm., Oxford, England). *Acta. Med. Scand.* 197(1/2):89-94, 1975.

Patients with ulcerative colitis or Crohn's disease were studied with respect to alterations in thyroxine metabolism. There were five patients with ulcerative colitis, of whom four had universal and one had distal involvement; and six patients with Crohn's disease, of whom three had extensive colonic disease, two widespread small bowel disease, and one ileocecal inflammation. Six of the 11 were severely ill and seven were treated with corticosteroids. None had evidence of thyroid disease. Eight healthy patients, of whom none were on any medication, served as controls. The thyroid was blocked by 240 mg potassium iodide given p.o. in two doses over 24 hr, and the blockade maintained by 60 mg twice daily throughout the experiment. One day after the start of the blockade, blood was drawn for T_4 estimation, immediately followed by i.v. injection of 10 μCi ^{131}I-T_4 in normal saline. Readings were made with a whole body counter once daily for six to ten days, considering the value 24 hr after administration as 100%. A thyroid count was taken 24 hr after injection and compared with a similar control count from the thigh. The daily retained radioactivity plotted semi-logarithmically produced a straight line. The thyroid count 24 hr after injection showed no appreciable amounts of the dose had been taken up by the thyroid. The patients had a significantly shorter ^{131}I-T_4 biological half-life and a higher daily fractional turnover than the controls. Severely ill patients had more pronounced changes than the mildly or moderately ill, or the controls. Corticosteroid treatment did not change either the half-life or the daily fractional turnover. Extrathyroidal T_4 iodine pools were not significantly smaller for the patients as a group than for the controls. Plasma T_4 and total T_4 iodine disposed/day did not differ significantly between patients and controls. It is concluded that patients with severe Crohn's disease or ulcerative colitis have increased daily fractional turnover of T_4.

5162 A STUDY OF THE CELLULAR INFILTRATE OF THE PROXIMAL JEJUNAL MUCOSA IN ULCERATIVE COLITIS AND CROHN'S DISEASE. *(E.)* Ferguson, R.; Allan, R. N.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Gut* 16(3):205-208, 1975.

The inflammatory cell infiltrate was studied from proximal jejunal biopsies of patients with ulcerative colitis, and patients with Crohn's disease. Of 20 ulcerative colitis patients, seven had active disease, six were on steroid therapy, eight had total colonic involvement, five had distal co-

lonic involvement, and seven had undergone panproctocolectomy. Of 26 patients with Crohn's disease, 16 had active disease, six were on steroids, four had known jejunal involvement, six had terminal ileal and colonic involvement, three colonic involvement only and four had ileal involvement only. For controls, 20 obese patients undergoing jejunoileal bypass were used. Biopsy specimens were taken from the first 10 cm of jejunum. Sections 5 μ thick were made and stained with hematoxylin and eosin. Cell counts were made in at least three areas of each section and the number of cells/mm^2 of lamina propria and cells/mm length of epithelium calculated. Plasma cells, lymphocytes, eosinophils, neutrophils, reticulum cells, or other cells in the lamina propria were counted. There were no differences in the lymphocyte counts in either tissue between any of the groups. However, patients with ulcerative colitis had higher lamina propria plasma cell counts (2268) than controls (1050) and Crohn's disease patients had higher counts (3002) than the ulcerative colitis patients. There were no significant differences between those with active or inactive disease, or those on steroid therapy and those not. In the ulcerative colitis patients, there were no significant differences between those with total colonic involvement and those with distal involvement, nor between all these patients and those who had previously undergone panproctocolectomy. In patients with Crohn's disease there were no differences between the various types of involvement. In this disease, the plasma cells outnumbered the lymphocytes in 15 biopsies. This was seen in only four ulcerative colitis patients, three of whom had active disease. It is concluded that raised plasma cell counts are present in ulcerative colitis and Crohn's disease, and may indicate an abnormal antigen in the proximal intestine.

5163 STRICTURE OF THE ANORECTUM IN CROHN'S DISEASE INVOLVING THE COLON. *(E.)* Greenstein, A. J.; Sachar, D. B.; Kark, A. E. (Mount Sinai Hosp., City Univ. New York, N.Y.). *Ann. Surg.* 181(2):207-212, 1975.

Twelve patients with anorectal stricture in a series of 160 patients with non-specific colitis (excluding ulcerative colitis) were studied. Of the 12, four had granulomatous colitis and eight had ileocolitis. Their ages ranged from 10-79 yr at the time of stricture development, but the mean duration from onset to the development of significant stricture was 14 yr. Nine of the 12 patients had strictures beginning 1/2 to 4 cm from the anal verge, three of which were short, fibrous, and diaphragmatic. The remaining five extended into the rectum for varying distances. Three of the 12 had strictures beginning 4, 5, and 7 cm from the anal verge and were either annular (under 2 cm) or tubular (over 2 cm), one of them extending to the rectosigmoid junction. Incontinence was found in three of the 12. Other pertinent clinical features were perianal disease (in 10), associated fibrous stricture or radiological narrowing (in 10) and discontinuity of the disease. Associated internal fistulae occurred in five and shortening of the bowel in three patients. Six had non-caseating granulomas. It is concluded that anorectal stricture occuring in Crohn's disease presents a major clinical problem, may lead to partial or complete obstruction, and may be associated with perianal disease, multiple fistulae, proximal stricturing in the bowel, and activity of Crohn's disease elsewhere.

5164 CHANGES IN THE BIDIRECTIONAL SODIUM FLUX ACROSS THE INTESTINAL MUCOSA IN CROHN'S DISEASE. *(E.)* Allan, R.; Steinberg, D. M.; Dixon, K.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Gut* 16(3):201-204, 1975.

Bidirectional sodium flux and net sodium absorption were compared in 10 patients with Crohn's disease and 11 patients with ulcerative colitis. All patients had been given a panproctocolectomy and ileostomy 1-15 yr previously, and were in good health with no radiological abnormality. After a 4 hr fast the patients were fitted with a nasogastric tube and a temporary ileostomy bag connected to a volume meter. Hartmann's solution (sodium 154 mEq/liter) was infused through the tube at 33 ml/min for 1 hr after an ileostomy output of 20-40 ml/min was established. Then 4 μCi ^{22}Na in 20 ml Hartmann's solution and 5 ml 1% BSP solution were given in a bolus *via* the tube, and the normal infusion of Hartmann's solution was continued for a second hr. The ileostomy output was measured by liquied scintillation counter in four 15 min aliquots and a fifth sample taken after the second hr. The last sample represented the concentration of label absorbed and resecreted from plasma to lumen, and was subtracted from the label absorbed and resecreted from plasma to lumen, which was subtracted from the label in the first four aliquots to give the fraction absorbed. The mean ileostomy output was significantly greater in the Crohn's disease patients than those with ulcerative colitis during the first hr but not during the second. The mean sodium flux both from lumen to plasma and from plasma to lumen was significantly lower in the Crohn's disease patients (3.30 and 2.30 mEq sodium/min, resp.) than in the ulcerative colitis patients (6.15 and 4.81 mEq sodium/min, resp.). There was no significant difference in the net absorption of sodium in the two groups. It is concluded that in Crohn's disease the gut may be more extensively involved than is shown by radiological appearance.

5165 THE THYROID IN ULCERATIVE COLITIS AND CROHN'S DISEASE. I. THYROID RADIOIODIDE UPTAKE AND URINARY IODINE EXCRETION. *(E.)* Jarnerot, G. (Med. Sch., Linkoping Univ., Sweden). *Acta Med. Scand.* 197(1/2):77-81, 1975.

Two studies were conducted to compare the prevalence of iodine depletion in chronic inflammatory bowel disease patients and healthy controls. The first study involved 50 patients, 10 of whom had distal and 10 universal ulcerative colitis; and 28 had Crohn's disease, of whom four had anorectal lesions, 11 more

widespread colonic lesions, and 13 small bowel lesions. Two patients had unclassified colitis. During the study 15 patients were given corticosteroids, and 32 Salazopyrin. All had 24 hr urine samples collected and analyzed for iodine excretion. Forty of the patients, of whom 15 were on a milk free diet, answered a dietary questionnaire, and were compared to 102 normal controls. The second study involved 38 patients of whom five had distal and nine universal ulcerative colitis, and 23 had Crohn's disease, of whom three had anorectal lesions, nine widespread colonic lesions, and 11 small bowel lesions. Thirty five were also included in the first study. Thirteen were given corticosteroids and 24 salicyclazosulfapyridine. Of the 35 patients in both studies, 12 were on corticosteroids. All second study patients had thyroid radioiodide uptake measured 24 hr after an oral test dose of 10 μCi ^{131}I. Of the 38 patients who answered a dietary questionnaire, 10 were on a milk-free diet. All were compared with a group of 36 healthy controls. The patient groups had considerably lower urinary iodine excretion levels and higher thyroid ^{131}I uptake levels than the corresponding control groups. All differences were significant except for radioiodide uptake in the males. Iodine excretion was significantly higher in the males than the females in both patient and control groups. Of the 50 patients in the first study, 10 had a 24 urinary iodine excretion of less than 40 μg, while five of the 102 controls did, a significant difference. Of 50 patients, 26 excreted less than 70 μg, compared to 28 of the 102 controls, a highly significant difference. Of the 38 patients in the second study, 16 had a 24-hr radioiodide uptake of 50% or more, while only four of the 36 controls had as great an uptake, a significant difference. Neither corticosteroid, salicylazosulfapyridine, nor milk-free diet had any effect on iodine excretion or iodide uptake. Patients not treated with corticosteroids, however, showed a highly significant negative correlation between excretion and uptake. There was no relation between excretion or uptake and the site of bowel disease. It is concluded that patients with chronic inflammatory bowel disease have increased thyroid uptake and diminished urinary iodine excretion, indicating iodine depletion.

5166 THE DISTRIBUTION OF MUCOSAL ANTIBODIES IN THE BOWEL OF PATIENTS WITH CROHN'S DISEASE. *(E.)* Green, F. H. Y.; Fox, H. (Dep. Pathol., Univ. Manchester, England). *Gut* 16(2):125-131, 1975.

The relative amounts of immunoglobulin-containing cells were studied in the affected gut areas of patients with Crohn's disease. Surgically resected unfixed specimens of large and small intestine were obtained from 24 patients. Blocks were taken from the resection lines, ulcerated areas, affected areas without ulceration, unaffected areas, and, when possible, attached mesenteric lymph nodes. Control blocks were taken from both large and small intestine from patients with carcinoma of the colon, and from normal peroral jejunal biopsies. The sections were fixed in acetone and stained directly or indirectly with anti-human IgA, IgM, IgG, antisera conjugated with fluorescein and isothiocyanate, and IgE antisera conjugated with fluorescein only. For controls, unstained sections were used to estimate autofluorescence. The identity of specifically fluorescent cells was checked by photographing them and restaining them with hematoxylin and eosin. In the Crohn's disease, the unaffected areas showed a slight increase in the IgA-containing cells of the lamina propria, while the epithelial IgA distribution was similar to that of controls. There was a definite increase in number of IgM-containing cells, to amounts equalling that of the IgA-containing cells, the epithelial fluorescence of the two showing a similar pattern in 19 papers. Distribution of IgG, IgD, and IgE-containing cells was similar to that of controls. Sections from abnormal but non-ulcerated areas showed a marked decrease in lamina propria IgA-containing cells. Intraepithelial IgA was reduced proportionately. IgM cell number was reduced relative to the unaffected areas but not to the controls. Epithelial IgM was largely absent. Cells containing other immunoglobulins did not differ significantly from controls or unaffected areas. In the ulcerated sections the inflammatory infiltrate contained moderate numbers of IgA, IgM, and IgG-containing cells in descending order. Lymphoid follicles containing predominantly IgA-containing cells were common in the submucosa, subserosa, and lymph nodes. The controls contained a predominance of IgA-containing cells in the plasma cells, and IgA in the epithelial cells. No epithelial fluorescence was seen when stained for other immunoglobulins. It is concluded that there is a focal reduction in IgA in the lamina propria and epithelium, confined to histologically abnormal areas in Crohn's disease, especially where the mucosa is still intact.

5167 FAT-REDUCED DIET IN THE SYMPTOMATIC TREATMENT OF PATIENTS WITH ILEOPATHY: A FOLLOW-UP STUDY. *(E.)* Andersson, H. (Sahlgrenska Hosp., Univ. Goteborg, Sweden). *Nutr. Metab.* 17(2):102-111, 1974.

Of 13 patients with chronic diarrhea who favorably responded to a low-fat diet in a previous clinical study, 10 were followed up for 3-27 months while continuing the diet. Of the 10, 9 had Crohn's disease and 7 of these had had an ileal resection. The 10th patient had had an extensive small bowel resection following a car accident. After the completion of the initial clinical study, the patients were given individually worked out diets adapted for use at home and work, containing about 40 g fat/day. All patients were given vitamin supplements, and one took cholestyramine during the study. Most patients were interviewed and given blood tests and routine examinations every 34 months. Daily intake of energy and nutrients was determined and bowel habits were registered. After the course of study the patients were readmitted to the clinic and given the same standard low-fat diet they were given in the initial clinical study. Nitrogen, potassium and sodium in feces, urinary calcium excretion, and total body potassium were determined. Most patients had few or no difficulties in following the diet. Mean decrease in energy intake between the periods before and after treatment was about 600 kcal/day. Fat intake was reduced from about 155 g to about 50 g/day. Protein intake was not affected except for an appreciable in-

crease in 2 patients. Bowel evacuations were reduced from 10-30/day before treatment to no more than 3/day after treatment, with 1 exception. In spite of the decreased caloric intake, body weight did not change significantly during the study. Total body potassium increased significantly in 5 patients. All patients showed low serum albumin, 2 abnormally so. In the clinic, following the at-home study, the daily number of stools and fecal excretion of fat, water, sodium, and nitrogen were all similar to values during the initial clinical period and the study period. It is concluded that a low-fat diet is beneficial in chronic diarrhea from ileal disease, even outside the hospital for a protracted period of time.

5168 CROHN'S DISEASE OF THE STOMACH: THE "RAM'S HORN" SIGN. *(E.)* Farman, J.; Faegenburg, D.; Dallemand, S.; Chen, C. K. (Downstate Med. Cent., Brooklyn, N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):242-251, 1975.

The radiological appearance of the stomach in 12 patients (9 men and 3 women; average age 30 yr) with Crohn's disease was studied. All had small bowel disease of varying extent and severity. Duodenal involvement was present in 5, colonic in 4, and esophageal in 1. In 9 of the 12, involvement of the antrum of the stomach produced a tubular shape with conical narrowing: the "ram's horn" configuration. In these cases the stomach distended poorly and peristalsis was sluggish and infrequent. In 2 other patients there was a pyloroduodenal deformity and in 1, irregular, abnormal coarse antral mucosal disease. Gastric dilation resulting from pyloroduodenal stenosis was present in 2 cases and excess retained gastric secretion in 3. The gastric mucosa ranged in appearance from coarsened and pebbly to smooth. It is concluded that gastric Crohn's disease can be diagnosed radiologically by the presence of a smooth tubular, poorly distensible antrum with sluggish peristalsis; cobblestone antral folds, moderate pyloroduodenal outlet obstruction, excess gastric secretions, and presence of Crohn's disease elsewhere in the gastrointestinal tract.

5169 REGIONAL ENTERITIS OF THE DUODENUM. *(E.)* Thompson, W. M.; Cockrill, H. Jr.; Rice, R. P. (Duke Univ. Med. Cent., Durham, N. C.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):252-261, 1975.

The characteristic roentgen features of duodenal regional enteritis were reviewed in 23 patients. Of the 23, 16 underwent surgery and presented with histological evidence of Crohn's disease. Diagnosis of Crohn's disease was also made by roentgenograms of the upper and lower gastrointestinal tract in each case. Clinically, 22 of the patients had abdominal pain and diarrhea, and 16 had vomiting and postprandial fullness. Seven were anemic and 16 had significant weight loss. In 6 patients the initial clinical diagnosis was peptic ulcer disease. Seven who showed duodenal changes had no upper gastrointestinal symptoms other than occasional pain. Roentgenographically, the appearance of the patients was similar to those with regional enteritis of the small intestine. There was concurrent gastric involvement in seven cases. The early stages of the disease showed a more subtle and less specific involvement than advanced regional enteritis. Findings included blunted, thickened, and possibly disorganized mucosa, and moderate narrowing of the lumen with some spasm. The most characteristic change in the late stages of the disease was the pseudopost Billroth I sign, in which the antrum, pylorus, and duodenal bulb are blended into a single tubular structure. Other late changes included loss of normal mucosal pattern and appearance of pseudodiverticula with ulceration, and occasionally a nodular appearance of the mucosa; a smooth, tapered stenosis sometimes resulting in obstruction, and sometimes involving the apex of the duodenal bulb; and duodenal fistulae, usually from the transverse colon to the second portion of the duodenum. It is concluded that Crohn's disease of the duodenum is more common than previously thought, and tubular narrowing of the antrum, pylorus, duodenal bulb, and second portion of the duodenum is diagnostic of the condition.

5170 TRANSPLANTATION ANTIGENS IN CROHN'S DISEASE: LINKAGE OF ASSOCIATED ANKYLOSING SPONDYLITIS WITH HL-AW 27. *(E.)* Russell, A. S.; Percy, J. S.; Schlaut, J.; Sartor, V. E.; Goodhart, J. M.; Sherbaniuk, R. W.; Kidd, E. G. (Dep. Med., Univ. Alberta, Edmonton, Canada). *Am. J. Dig. Dis.* 20(4):359-361, 1975.

The HL-A antigen profile was studied in 77 patients with Crohn's disease to determine if any significant linkage exists between a genetic susceptibility to this disease and any of the transplantation antigens. A microcytotoxicity technique was used to determine the complete HL-A antigen profile in the Crohn's disease patients, 11 of whom had ankylosing spondylitis, and 600 controls. The profiles did not differ significantly between patients and controls. However, among the 11 males with ankylosing spondylitis, 10 patients were positive for HL-Aw 27; the frequency of this antigen in this series resembled that found in spontaneous ankylosing spondylitis. Among the other 66 Crohn's disease patients, three females had HL-Aw 27 with no evidence of sacroiliitis. In seven patients with Crohn's disease and associated spondylitis, the spondylitis preceded Crohn's disease by 1.5-10 yr, while in two patients the diseases appeared at approximately the same time and in two others Crohn's disease preceeded the spondylitis by 0.5-3 yr. The results suggest that the HL-Aw 27 antigen may be a useful indicator of spondylitis in Crohn's disease.

5171 CLINICAL PATTERNS IN CROHN'S DISEASE: A STATISTICAL STUDY OF 615 CASES. *(E.)* Farmer, R. G.; Hawk, W. A.; Turnbull, Jr., R. B. (Cleveland Clin. Found., Ohio). *Gastroenterology* 68(4): 627-635, 1975.

Clinical patterns were studied in 615 cases of Crohn's disease to determine whether initial anatomic involvement in this disease is directly related to the clinical course and prognosis. The patients repre-

sented 615 consecutive cases of Crohn's disease seen between 1966 and 1969. All data concerning these patients were subjected to statistical analysis based on determinations of anatomic involvement, clinical symptoms, course of the illness as shown by follow-up data, and surgical experience. The following initial clinical patterns were established: ileocolic pattern with involvement of the distal ileum and right colon (252 patients); small intestinal pattern with involvement of the small intestine but not the colon (176); colon pattern with colonic but not small intestinal involvement (114); and anorectal pattern with anorectal but not proximal colonic or small intestinal involvement (21). Statistically significant symptoms included rectal bleeding with colon involvement, and rectal fistulas with ileocolic or colon involvement. The statistically significant complications included: perianal fistulas, internal fistulas, and intestinal obstruction with the ileocolic pattern; perianal fistulas, toxic megacolon, and arthritis with the colon pattern; and intestinal obstruction with the small intestinal pattern. Surgery was required in 73% of the ileocolic pattern patients and 51% of the small intestinal and colon pattern patients. Anorectal involvement was rarely the initial manifestation, but frequently accompanied the ileocolic and colon patterns. There were postoperative recurrences in 27% of the patients with the ileocolic pattern, 21% of those with the small intestinal pattern, and 16% of those with the colon pattern. Death occurred in 7% of the ileocolic pattern patients, 5% of those with the colon pattern, and 4% of those with the small intestinal pattern. The data indicate that Crohn's disease should be considered as a protean disease with certain definable patterns and that anatomic involvement at the time of diagnosis bears directly on the clinical course, type of complications, and prognosis.

5172 REMEDIABLE DEFECTS IN CROHN DISEASE. A PROSPECTIVE STUDY OF 63 PATIENTS. *(E.)*
Beeken, W. L. (Dep. Med., Univ. Vermont, Burlington). *Arch. Intern. Med.* 135(5):686-690, 1975.

To identify potentially remediable abnormalities in Crohn's disease, the incidence of anemia, serum electrolyte deficiencies, defects in carbohydrate, fat, and vitamin B_{12} absorption, jejunal bacterial overgrowth, and abnormalities in intestinal protein metabolism were prospectively analyzed in 63 patients with this disorder. Radiological studies showed disease in the upper part of the small bowel in 14% of the patients, the mid-small bowel in 22%, the lower part of the small bowel in 76%, and the colon in 33%. Hematocrit values were less than 40 volume percent in 67% of the patients, and iron deficiency was found in 35%. In addition, 10 patients had low serum folate values and conventional Schilling tests with intrinsic factor were abnormal in 31 patients. Of the entire group, 44 patients had one or more potentially correctable causes of anemia; 21 had serum electrolyte abnormalities, the most common of which was hypocalcemia; 13 had abnormal lactose tolerance tests, and jejunal disease or bacterial overgrowth were possible contributing factors in six. Xylose malabsorption did not accurately reflect jejunal disease. All patients showed normal hydration and renal function. Of the 61 patients who completed fat balance testing, 19 had steatorrhea caused by a variety of factors. Derangements of protein metabolism were the most commonly encountered abnormalities: excessive enteric protein loss was present in 76%; fasting serum tryptophan values were low in 29 or 35 patients tested; fecal nitrogen and nitrogen balances were abnormal in most cases; and hypoalbuminemia was present in 16 patients. High bacterial colony counts were found in 30% of the jejunal cultures. In addition to the initial barium studies, Crohn's disease patients should be evaluated with regard to body weight, stool frequency, and anemia. A selective search for correctable abnormalities will often identify defects amenable to correction by appropriate replacement therapy.

5173 PRESTENOTIC ENTERITIS AND ENTEROCOLITIS IN CHILDREN: DESCRIPTION OF A SYNDROME AND REVIEW OF FIVE CASES. *(E.)* Smith, E. I.; Gill, C. C. (Child. Mem. Hosp., Univ. Oklahoma Coll. Med., Oklahoma City). *South. Med. J.* 68(4): 427-430, 1975.

Case reports of five children with enteritis proximal to a stenotic area of the bowel are presented. The first patient was born with multiple congenital anomalies including a first arch syndrome and a low imperforate anus with a perineal fistula; she had perineal anoplasty on the seventh day of life. The second patient was a microcephalic male born with an imperforate anus; he had abdominoerineal anoplasty on the second day of life. The third patient (female) was born with multiple congenital anomalies including a high imperforate anus and had a colostomy performed soon after birth. The fourth case was a female born with a caudal duplication abnormality and anal atresia; she was subjected to numerous surgical procedures including a transverse colostomy as a neonate. The last case was a boy born with multiple atretic areas of the small intestine; intestinal obstruction was diagnosed at 72 hr of age. A clinical syndrome involving prestenotic enteritis and enterocolitis consisting of abdominal distention and pain, intermittent diarrhea and constipation, failure to thrive, fever, and, in some patients, extreme prostration and death was observed. The pathogenesis of this enteritis and enterocolitis was apparently related to partial mechanical obstruction of the bowel with proximal dilatation, stagnation, and capillary stasis. Acute relief from the enteritis and enterocolitis was obtained by stomal dilatation and colonic irrigation, but all patients eventually required surgical intervention. Because mortality in this disease is significant and the results of early surgical intervention are favorable, surgical intervention is encouraged at the first sign of prestenotic enteritis or enterocolitis in children. Surgical correction of the distal stenosis produced a growth spurt in the two successfully treated patients in this series without other growth-limiting diseases.

5174 DIAGNOSIS OF CROHN'S RECURRENCE AFTER SURGERY. *(E.)* Calvadetscher, P.; Deyhle, P. (Dep. Intern. Med. Univ. Zurich, Switzerland). *Endoscopy* 7(1):27-29, 1975.

Coloscopy was compared with clinical observation and radiology in diagnosis of recurrent Crohn's disease at or near the site of earlier surgery. Studies were done on 12 patients; the average interval between radiological and endoscopic examination was 13 days; the longest interval was 67 days. Recurrence was defined clinically as return of symptoms after a postoperative symptomless period; radiologically as appearance of spiculas, cobblestone-pattern and restriction of the lumen; and endoscopically as ulcerations and cobblestone pattern. Endoscopy showed recurrence in 11 patients, while radiology showed recurrence in only 3. An ulcer at the anastomosis was shown in one case by radiology, in contrast to 6 cases by endoscopy. Cobblestone patterns were found by radiology in 3 of 5 patients. They produced clinical symptoms in 4. Clinical symptoms developed in 3 cases of anastomotic ulcers, undetected radiologically. It is concluded that endoscopy is superior to radiology in diagnosis of these recurrences, and can detect recurrences during symptomless phases.

5175 VARIOUS MANIFESTATIONS OF REGIONAL ENTERITIS AND ITS DIFFERENTIAL DIAGNOSIS BY RADIOLOGY. *(Ger.)* Koischwitz, D.; Sobbe, A.; Schirmer, G. (Radiol. Clin., Univ. Bonn, Germany). *Roentgenblaetter* 28(1):36-47, 1975.

5176 TWO FIRST DEGREE RELATIVES WITH DILATATION OF THE COLON DUE TO CROHN'S DISEASE. *(E.)* Mallinson, C. N.; Candy, J. C.; Cowan, R.; Prior, A. (Greenwich Hosp., England). *Guys Hosp. Rep.* 122 (384):211-220, 1973.

5177 CROHN'S DISEASE OF THE COLON. III. TOXIC DILATATION OF THE COLON IN CROHN'S DISEASE. *(E.)* Greenstein, A. J.; Kark, A. E.; Dreiling, D. A. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 63(2):117-128, 1975.

5178 EPIDEMIOLOGICAL ASPECTS OF CROHN'S DISEASE IN CLYESDALE 1961-1970. *(E.)* Smith, I. S.; Young, S.; Gillespie, G.; O'Connor, J.; Bell, J. R. (Stobhill Gen. Hosp., Glasgow, Scotland). *Gut* 16(1):62-67, 1975.

5179 CROHN'S DISEASE OF THE COLON. II. CONTROVERSIAL ASPECTS OF HEMORRHAGE, ANEMIA AND RECTAL INVOLVEMENT IN GRANULOMATOUS DISEASE INVOLVING THE COLON. *(E.)* Greenstein, A. J.; Kark, A. E.; Dreiling, D. A. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 63(1):40-48, 1975.

5180 MALIGNANT TUMORS ASSOCIATED WITH GRANULOMATOUS ENTEROCOLITIS. *(E.)* Kim, U.; Aufses, Jr., A. H.; Kreel, L. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 63(1):66-70, 1975.

5181 RECTAL BIOPSIES FROM THE BY-PASSED RECTUM. *(E.)* Thompson, H. (Dep. Pathol., Univ. Birmingham, England). *Arch. Fr. Mal. App. Dig.* 63 (7):583-584, 1974.

5182 ILEO-RECTAL ANASTOMOSIS IN CROHN'S DISEASE. *(E.)* Alexander-Williams, J.; Steinberg, D. M.; Allen, R. N.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Arch. Fr. Mal. App. Dig.* 63(7):588, 1974.

5183 CROHN'S DISEASE. *(Fr.)* Soussaline, M. (no affil.). *Rev. Fr. Gastroenterol.* (99): 39-42, 45-46, 50, 1974.

5184 CELLULAR IMMUNE REACTION TO COMMON ANTIGEN AND HUMAN SMALL AND LARGE INTESTINE ANTIGENS IN PATIENTS WITH REGIONAL ENTERITIS, ULCERATIVE COLITIS, AND CIRRHOSIS. *(Ger.)* Eckhardt, R.; Heinisch, M.; Meyer zum Buschenfelde, K. H. (2nd Med. Clin., Univ. Mainz, Germany). *Z. Gastroenterol.* 12(Suppl.):431-432, 1974.

5185 ENDOSCOPIC FEATURES OF CROHN'S DISEASE IN THE ESOPHAGUS, STOMACH, DUODENUM, JEJUNUM, ILEUM, AND COLON. *(Ger.)* Fruhmorgen, P.; Rosch, W. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Z. Gastroenterol.* 12(8):592, 1974.

5186 CROHN'S DISEASE OF THE COLON. *(Ger.)* Hammer, B.; Engelhart, G.; Hugel, H.; Gloor, F. (Cantonol Hosp., St. Gallen, Switzerland). *Z. Gastroenterol.* 12(8):592-593, 1974.

5187 SEVERE ACUTE RECURRENT GASTROINTESTINAL BLEEDING IN CROHN'S DISEASE. *(Ger.)* Keller, M.; Scheurer, U.; Wanger, F.; Voegeli, E.; Halter, F. (Inselsp., Bern, Switzerland). *Z. Gastroenterol.* 12(8):595, 1974.

5188 FOLLOW-UP STUDIES OF CROHN'S DISEASE DURING AZATHIOPRINE TREATMENT. *(Ger.)* Fehr, H. F. (Aarau Cantonol Hosp., Switzerland). *Z. Gastroenterol.* 12(8):595-596, 1974.

5189 ENDOSCOPIC DIAGNOSIS OF RECURRENCES AFTER SURGICAL TREATMENT OF CROHN'S DISEASE OF THE COLON AND ILEUM. *(E.)* Clavadetscher, P.; Deyhle, P. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(8):593-594, 1974.

5190 ANATOMICAL AND CLINICAL FORMS OF CROHN'S DISEASE AND THE RESULTS OF SURGERY IN 13 CASES. *(Fr.)* Saubier, E. C.; Brault, A.; Partensky, C. (Edouard Herriot Hosp., Lyon, France). *Lyon Chir.* 70(5):289-293, 1974.

5191 MAGNESIUM METABOLISM IN PATIENTS WITH CHRONIC INFLAMMATORY DISEASE OF THE BOWEL. *(E.)* LaSala, M. A.; Lifshitz, F.; Pergolizzi, R.; Silverberg, M.; Wapnir, R. A. (North Shore Univ. Hosp., Manhasset, N.Y.). *Am. J. Clin. Nutr.* 28(4): 442, 1975.

5192 CROHN'S DISEASE: ANAL LESIONS. *(E.)* Lockhart-Mummery, H. E. (no affil.). *Dis. Colon Rectum* 18(3):200-202, 1975.

5193 CROHN'S DISEASE: ASSOCIATED DISEASES. *(E.)* Schofield, P. F. (no affil.). *Dis. Colon Rectum* 18(3):192-193, 1975.

5194 CROHN'S DISEASE: RECOGNITION IN THE OPERATING ROOM. *(E.)* Weakley, F. L. (Cleveland Clin., Ohio). *Dis. Colon Rectum* 18(3):198-199, 1975.

5195 CROHN'S DISEASE: MEDICAL MANAGEMENT. *(E.)* Cooke, W. T. (Gen. Hosp., Birmingham, England). *Dis. Colon Rectum* 18(3):194-197, 1975.

5196 CROHN'S DISEASE: PRECANCEROUS CHANGES IN COLITIS. *(E.)* Morson, B. (St. Mark's Hosp., London, England). *Dis. Colon Rectum* 18(3): 207-208, 1975.

See also, 4665, 4704, 4719, 4720, 5131.

NUMBERS FOLLOWED BY AN ASTERISK INDICATE ABSTRACTS. OTHER NUMBERS REFER TO CITATIONS.

ABAD, J.
4892
ABBRUZZESE, A
4199*
ABE, Y.
4500*
ABENDROTH, K.
4843
ABENGOWE, C.U.
4943*
ABTAHI, F.S.
3929*
ABUPANTANI, P.
4228
ABUPATANI, R.
4223, 4233, 4252
ACCARY, J.P.
3963, 5025
ACKAMNN, J.J.
4784*
ACKERMAN, H.W.
4878
ADAIKAN, G.P.
3967
ADAMI, G. F.
4939
ADAMIAK, T.
4930
ADAMIS, E.
5030
ADAMSONS, R.J.
4040
ADAMUS, J.
4462
ADLOFF, M.
4769
AEBERHARD, P.
3883
AENISHANSLIN, H.W.
4433
AETEI, E.
4919*
AESCHLIET, M.
4869
AGAFONOVA, N.Y.
4075
AGARWAL, H.N.
4375*
AGRAWAL B.
5154
AGRAWAL, R.M.
4293, 4296, 5130
AGUIRRE, M.
4792*
AHONEN, A.
4164*
AIKAT, M.
4705
AKEDA, S.
4863, 4864
AKITA, H.
4957
AL-HUMADI, A.H.
4702
AL-NAGDY, S.A.
5087
AL-REBATE, I.
4608*
ALAAMA, A.
4475
ALAGILLE, D.
4785*
ALARIF, A.
3993
ALBINUS, M.
3944*
ALEXANDER-WILLAIMS, J.
4484*
ALEXANDER-WILLIAMS, J.
4219, 4517, 5182
ALEXIOU, D.
4768
ALFORD, J.E.
4702
ALI, S.S.
4128
ALIMOVA, E.K.
4111
ALLAN, R.
5164*
ALLAN, R.N.
5162*
ALLARD, M.
4832
ALLEN, R.N.
5182
ALONSO, D.
3930*
ALPERT, E.
4871*
ALRENGA, D.P.
4783*
ALVES, M.A.
4360
AMENT, M.
4291
AMENT, M.E.
4712
AMERIKS, J.A.
4790*
AMMANN, R.
4395, 4997
AMPE, J.
4966
AMSTERDAM, A.
3976*
ANDERSON, C.M.
4602*, 4632, 4656*
ANDERSON, M.C.
4165*
ANDERSON, S.
3966
ANDERSSON, H.
5167*
ANDES, W.A.
4390*
ANDREASEN, P.B.
3996*
ANDREIMAN, L.A.
4035
ANDRESEN, B.R.
4912*
ANDREUZZI, P.
4923*
ANDREWS, W.H.H.
4100
ANDROULAKIS, G.
4168*
ANDROULAKIS, J.
4168*
ANFOSSI, A.
4039
ANISIMOWICZ, L.
4967
ANITA, F.P.
4504*
ANTHONY, P.P.
4794*
APARICO, N.J.
3957
APENTTILA, A.
4164*
APPELMAN, H.D.
4790*
APPERT, H.E.
3906
ARBEITER, C.
4928
ARBEITER, G.
4261
ARCERITO, S.
4587
ARCHIMANDRITIS, A.
4664*
ARCIELLO, G.
4846
ARENDT, R.
4787*
ARGLEBE, C.
3913
ARIANOFF, A.A.
4625
ARIAS, I.M.
4181
ARIF, S.
4040
ARIMA, Y.
4957
ARMANINI, G.B.
4319
ARNDORFER, R.C.
3873*, 4299
ARNESJO, B.
4738*, 4950*
ARNOLD, A.
4080
ARNOLD, W.
4880
AROLD, R.
3913
ARONSON, A.S.
4556
ARSENAULT, A.
4374*
ARYA, R.K.
4380*
ASHMORE, J.
4051
ASHREFOV, A.A.
4976
ASQUITH, P.
4484*, 4640
ASSOR, D.
4344

ASTE, H.
4590
ATALLAH, P.
4569
ATLAS, H.
4932
ATWELL, D.T.
5022
AUBERT, P.
4564
AUDIBERT MENA, L.
4700
AUFSES, A.H., JR.
5180
AUGER, C.
4937
AUMAITRE, A.
3814*
AUST, J.B.
5081
AVANT, G.R.
4793*
AVILA, F.
5042
AVILOVA, O.M.
4303
AYLETT, S.G.
4718
AYNEDJIAN, H.S.
4006*
AYOOLA, E.
4294
BABALA, J.
4746
BABICH, A.
4040
BABICHEV, S.
4448
BACCIU, P.P.
4587
BACHOFEN, H.
4191*
BACK, G.
3836
BACK, P.
4969
BACKER, O.G.
4967
BACQUE, P.
4470
BADALAMENTI, J.
4501*
BADER, G.
4774
BAERLOCHER, C.
4424
BAGRATIONI, E.L.
4028
BAIBEKOVA, E.M.
4450
BAILEY, B.A.
4491*
BAILEY, D.T.
3804
BAILEY, W.
4649*
BAKER, S.J.
4172
BAKER, W.N.W
4721
BAKI, A.
5063
BALESTRAZZI, A.
4452, 5064
BALESTRAZZI, N.
4452
BALESTRIERI, G.G.
4846
BALEVSKA, P.S.
4082
BALTHAZAR, E.J.
4290, 4696
BANA, J.
4660
BANCHERI, C.
4828
BANK, N.
4006*
BANK, S.
3958
BAQUERO, F.
5082
BAR, W.
4404
BARAG, N.
4309, 4313
BARANOVA, K.F.
4241
BARANY, F.
4711*
BARAONA, E.
4154*
BARASHKOVA, G.M.
3882
BARBA, F.I.
4889
BARBA, G.
4868
BARBER, D.C.
4526
BARBEZAT, G.O.
3958
BARBIER, J.P.
4449
BARBIROLI, B.
4021
BARCHUNOV, B.N.
4201
BARCHUNOVA, N.M.
4201
BARDHAN, K.D.
4557
BARISHEVA, L.N.
4201
BARLOW, B.
5017*
BARNABEI, O.
3995*
BARNSLEY, W.
4342
BARON, J.H.
3945*, 4219
BARRET, C.
4269
BARRETT, G.S.
3909
BARROS D'SA, A.A.J.
3945*
BARTELHEIMER, W.
4203, 5043
BARTOLOZZI, G.
4856
BARTRUM, R.J. JR.
4199*
BARTSCH, G.
4104
BASOLI, A.
4946*
BATONYI, E.
4461
BATTISTINI, C.
5006
BAU, J.
4838
BAUER, P.
3941*
BAULIEUX, J.
4024
BAUMGARTNER, I.
4910
BAUMGARTNER, R.
4426
BAYARD, F.
5067
BAYER, P.M.
4190*
BAYFIELD, R.F.
4048
BAYLESS, T.M.
4644
BEAHRS, O.H.
5111
BEAL, J.M.
4675
BEAUFIL, B.
4775
BEAUGIE, J.M.
4768
BEC, P.
5067
BECHER, R.
4571
BECK, A.R.
4186*
BEDI, B.S.
3883
BEDINE, M.S.
4644
BEEKEN, W.L.
5172*
BEEVERS, D.G.
4663
BEGER, H.G.
3888
BEGIC-JANEV, A.
5139
BEHER, W.T.
4133
BEIER, L.V.
4231
BEKAERT, S.
4869

BEKHTI, A.
5149
BEKTI, A.
5143
BELANGER, G.
4915
BELASCU, M.
5028
BELESLIN, D.B.
3897
BELL, J.R.
5178
BELL, J.U.
4036
BELOV, I.N.
4211
BELSITO, A.A.
4287, 4288
BENVENUTI, G.A.
4289
BEN BOUALI, A.
4302
BENCUR, J.
4746
BENEDEK, S.
4205
BENEDETTI-VALENTINI, G.
4847
BENEDETTI-VALENTINI, S.
4847
BENINTENDI, V.
5005
BENKEN, U.
4532
BENNETT, A.
3903
BENNETT, C.E.
5158
BENYSEK, L.
4591
BERARD, A.
4155*
BERARD, J.
4024
BERARD, P.
4024
BERCI, G.
4272
BERGER, H.G.
4098
BERGER, R.
4523
BERGIEL, A.
4906*, 4925*
BERNADES, P.
5025, 5115
BERNARD, W.
4341
BERNDT, H.
4305, 4354
BERNER, P.
3941*
BERNIER, J.J.
3839
BERNIER, L.
4561
BERQUIST, T.H.
4200*
BERRY, B.E.
4340
BERRY, D.E.
4081
BERRY, L.J.
4066
BERTELSEN, S.
4362
BERTHELOT, P.
4798
BERTHILLIER, G.
4093
BERTILLIER, G.
4007*
BERTRAM, P.D.
4477
BERTRAND, J.
4521
BERTRAND, M.B.
4561
BESSER, G.M.
3944*
BETTARELLO, A.
4651*, 4935
BEYRISS, K.
4608*
BEZARD, J.
3791*
BHAT, R.V.
4861*
BHATTA, A.D.
4756
BHISE, K.B.
4507*
BHIWANKAR, N.T.
4499*
BIAGOVIDOV, D.F.
4762
BIANCA, T.
3902
BIANCO, A.
4895
BIANCO, S.
4936
BIASIATO, R.
4357
BIBER, H.D.
4078
BICZYSKO, W.
4908
BIEDERMAN, M.A.
4361
BIELECKI, M.
4671
BIGOT, J.M.
4567
BILLINGS, J.A.
3899
BILLINGS, R.E.
4051
BILSKI, R.
3978*
BILYK, I.
4263
BINDER, R.J.
4311
BINDER, S.C.
4717
BINI, A.
4062
BINSWANGER, U.
4779, 4997
BIRHRER, R.
4997
BIRIULINA, T.I.
4823
BISHOP, R.F.
5014*
BISMUTH, H.
4942*, 4954*
BITTNER, R.
4098
BIVINS, B.A.
4244
BLACK, N.B.
4084
BLACKSTONE, M.O.
4476
BLACOMBE, M.
4775
BLAIR, E.L.
3973
BLAIR, J.A.
4637
BLANCHARD, J.
4994
BLAZEK, G.
4821
BLESOVSKY, A.
4342
BLICHERT-TOFT, M.
4362
BLOCH, R.
4628
BLOCK, M.A.
4953*
BLOOM, A.L.
4835
BLOOM, S.A.
3945*
BLOOM, S.R.
4490*
BLUM, A.L.
3889, 4339
BLUM A.L.
4423
BLUM, A.L.
4515, 4659
BLUMGART, L.H.
4105, 4835
BOBBIO, P.
4565
BOBKOVA, S.D.
5098
BOBOIS, J.P.
4959
BOCCATO, P.
4121
BOCKEL, R.
4518
BOCZ, M.
4971
BODDAERT, J.
4692
BODE, J.C.
4247, 4806

BODEN, G.
3937*
BODIN, F.
4393
BODVALL, B.
4950*
BOEY, J.
4650*
BOGDANOV, G.N.
4023
BOGNACKI, J.
3854
BOGUSLAWSKI, M.
4159
BOHDAN, I.P.
4089
BOIADZHIEVA, Z.H.K.
4041
BOIKO, M.S.
4889
BOISSON, M.
4577, 4586
BOKERIIA, R.I.
4028
BOLOGNA, A.
4558
BON, R.
4894
BONDAR, Z.A.
4761
BONDARENKO, G.V.
4974
BONFILS, S.
5025, 5115
BOND, J.A.
4209
BONOLA, G.
4070
BONTING, S.L.
3949
BOOTH, R.A.D.
3924*
BORDES, M.
4178
BOREL, G.A.
5059
BOREUS, L.O.
4854*
BORGIEL, W.
4968
BORIAN, A.
5030
BORNSCHEIN, W.
4253
BORRELLI, A.
4903*
BOSCHI, J.
4207
BOSSECKERT, H.
4843
BOULARD, C.
5067
BOUSTANY, F.N.
4569
BOUTOVITCH, N.
4994
BOVEY, F.A.
4174
BOWN, R.L.
4654*
BOYER, K.M.
5112
BRACALE, G.C.
4836
BRADBROOK, R.A.
4105
BRADLEY, D.J.
4100
BRADLEY, V.D.
4295
BRANDAO, M.S.
4737*
BRANDT, C.
4518
BRANDT, P.
4962
BRARD, E.
3901
BRASHEAR, H.R.
3804
BRASSINNE, A.
5024
BRAUER, D.
4098
BRAULT, A.
5190
BREINING, H.
4408
BREMMELGAARD, A.
3996*
BRESSAN, G.C.
4991
BRET, P.
4577, 4586
BRICARD, H.
5054
BRINKMAN, W.F.B.
4216
BRISKIN, B.S.
4448
BROCHERIOU, C.
5063
BRODANOVA, M.
4888
BRODERSON, M.
4813
BRODMERKEL, G.J., JR.
4293, 4296, 5130
BROICHER, K.
4350
BROMBART, M.
4243
BRON, B.A.
4339, 4423
BROOKS, J.
4187*
BROUWERS, H.P.
3955
BROWN, B.N.
4530
BROWN, C.
4188*
BROWN, G.
4893
BRUCKNER, H.W.
4657*
BRUGGEMANN, J.
5091
BRUGMANN, E.
4260
BRUGO, E.A.
4554
BRUN, A.
4556
BRUN, D
4915
BRUNNER, H.
4916, 5002
BRUNT, P.W.
4485*
BRUSCHKE, G.
4773
BRUSH, B.E.
4953*
BRYK, D.
4373*
BRYNES, R.K.
4648*
BUCHHOLZ, K.
4611*
BUCZOWSKI, N.
5046
BUDAKOV, P.
4266
BUDILLON, G.
3969
BUKIN, IU.V.
4091
BULETSA, V.I.
3960
BULKINA, G.I.
4974
BUNIN K.V.
5040
BUNTROCK, P.
4844
BUONGIORONO, R.
4897
BUR, F.
4768
BURELLE, H.
4577
BURGER, K.
4933, 4939
BURKE, M.D.
4292
BURKITT, D.P.
5089
BURLACHENKO, V.P.
4981, 4990
BURLINA, A.
4121
BURRELL, C.R.
4873*
BURRINGTON, J.D.
4829, 5084
BURROWS, F.G.D.
4630
BURUCUA, J.E.
4926
BUSCH, H.
4019
BUSINCO, E.
5038

BUSSE, R.
4203
BUSSMANN, J.F.
4588
BUTCHART, E.G.
4342
BUTCHER, F.R.
3912, 4080
BUTLER, D.P.
4047
BUTSCH, J.L.
4702
BUTT, K.
4040
BUTTERWORTH, K.R.
3917
BUTTI, A.
4895, 4936
CADILLI, G.
4868
CADY, B.
4781*
CAGAN, E.M.
4333
CAGOSSI, M.
4936
CALAHANE, S.F.
5080
CALAME, K.B.
3974*
CALDER, J.F.
4945*
CALHOUN, W.K.
4047
CALVADETSCHER, P.
5174*
CAMERON, J.L.
4739*, 4940*
CAMPRODON, R.
4600
CANDY, J.C.
5176
CANEDO, T.
5082
CANEPA, M.
4840
CANTONI, P.
3975*
CANO, R.
4290
CANTONI, L.
4923*
CANTY, T.
4576
CANZLER, H.
5041
CAPPON, I.D.
4068
CARBONI, M.
4946*
CARDIN, C.J.
3848
CARLIER, H.
3791*
CARLSON, G.P.
4011*
CARLSON, H.C.
4200*
CARNEY, C.N.
4298
CARNOT, F.
4449
CAROLLO, F.
4868
CARRELLA, M.
3969
CASANOVA, P.
4610*
CASPAR, J.
4784*
CASPARY, W.F.
3833, 3837, 5026
CASTALDO, M.
4846
CASTEELS-VAN DAELE, M.
4865
CASTELL, D.O.
4486*
CASTELLANOS, J.
4740*
CASU, A.
4118, 4119
CATALANO, D.
4225
CATHELINEAU, L.
4920*
CATTERALL, M.
4441
CAVALIERE, P.
4039
CEISLINSKA, K.
4714
CELLI, L.
4227
CERF, M.
4881
CESSI, C.
4062
CEVESE, P.G.
4357
CHABANON, R.
4239, 4676
CHACAJ, W.
4714
CHACIN, J.
3930*
CHACON, J.P.
4527
CHAFFEE, V.
4195*
CHAMBON, J.
3984
CHAMLIAN A.
4610*
CHANARIN, I.
4913*
CHANDRASEKARAN, S.
4293, 4296
CHANG, Y.C.
4199*
CHANH, P.H.
3896
CHARLAS, J.
4855
CHASSEAUD, L.F.
4059
CHEDEKEL, M.
4174
CHEDID, A.
3790*
CHELAB, A.
4520
CHELI, R.
4590
CHELIDZE, P.V.
4143
CHEN, C.K.
5168*
CHEN, M.L.C.
5111
CHEN, T.H.
4815
CHERET, A.M.
3947
CHERIATHUNDAM, E.
3974*
CHERMET, J.
4567
CHERNOUSOV, A.F.
4445
CHERNUKHINA, L.D.
4113
CHERUBINI, M.
4038, 4136
CHEVAN, J.
3810*
CHIAMENTI, C.
4818
CHIANG, S.P.
4758
CHIARAMONTE, M.
4903*
CHICHE, B.
4402
CHIGOT, J.P.
4535
CHIKARAISHI, T.
4411
CHILIMONIUK, M.
4596
CHILLA, R.
3913
CHIMOMBE, H.E.
4246
CHINAGLIA, A.
4420
CHISHOLM, D.M.
5124
CHKEIDZE, R.D.
4579
CHOI, B.H.
4065
CHOJNACKI, J.
4930
CHONG, G.C.
5111
CHORVAT, V.
4746
CHORVATHOVA, V.
4046
CHOU, A.C.
3925*
CHRISTANSEN, J.
4498*

CHRISTEN, H.
4838
CHRISTENSEN, J.
3876*
CHRISTIANSEN, L.A.
4362
CHRISTIE, D.L.
4291
CHUA, K.L.
4268
CHURAR, S.V.
4267
CHUTTANI, H.K.
4375*
CHUVILSKAIA, L.M.
4175
CHVAPIL, M.
4057
CIAMPOLINI, M.
4856
CIANCAMERLA, G.
4590
CIESLAKOWA, D.
3824, 4153*
CIESZKOWSKI, M.
3931*
CIMINO, L.
3969
CINCALA, R.P.
4293, 4296, 5130
CIZNAR, I.
5107
CLARK, M.L.
4605*, 4654*
CLARK, S.B.
4161
CLASSEN, M.
4248, 4258
CLAUSEN, E.
4791*
CLAVADETSCHER, P.
4684, 4997, 5189
CLEMENT, J.
3850
CLINE, W.S.
4473
CLODI, P.H.
3829
COBET, G.
5103
COCHRAN, K.M.
4376*
COCKRILL, H., JR.
5169*
COENE, J.A.
4572
COHEN, S.
3905
COLCHER, H.
4360
COLEMAN, R.
4003*
COLIN J.
4855
COLMAN, M.
4940*
COLOTORTI, E.A.
4193*
COLTORTI, M.
4116, 4812
COLY, D.
5147
COMBES, B.
4875*
COMSTOCK, G.W.
4859*
CONKLIN, J.L.
3876*
CONLON, T.P.
3817*
CONNEY, A.H.
4083
CONNOR, A,M.
4185
CONSTANTIN-VOILLEMOT, N.
3798, 3799
CONTE, V.P.
4935
COOKE, W.T.
5162*, 5164*, 5182, 5195
COOPER, W.
3831
COOPER, W.D.
3911
COPE, V.
4284
COPELAND, E.M.
3868*
COPPLESON, L.W.
4648*
CORALLE, J.
4189*
CORBEEL, L.
4865
CORBETT, R.L.
3804
CORBIC, M.
5025
CORCINO, J.J.
4623
CORDIANO, C.
4357
CORDIOLI, G.P.
3971
CORDOBA, I.
4329
CORKERY, J.J.
4656*
CORLETTE, M.B.
4954*
CORNET, A.
4449
CORNFIELD, D.
3930*
CORRODI, P.
4779
CORSI, M.
4279
COSTANZI, J.
4554
COSTOPOULOS, L.B.
4707
COUGARD, A.
4610*
COUSINS, R.J.
4069
COWAN, R.
5176
COY, D.H.
3944*
CREMA, A.
3863*
CRET, R.
4024
CREUTZFELDT, W.
4759, 5039
CRICHLOW, R.W.
4235
CRISTAU, P.
4894
CROOM, R.D. III
4210
CRUMIERE, C.
4855
CTETSYUK, A.G.
4031
CULEBRAS, J.
4187*
CUMMINGS, J.H.
3840, 3899
CURR, S.
4923*
CURRARINO, G.
4365
CUSSEN, L.J.
5021
CUSTER, H.R.
4356
CZAJA, A.J.
4390*
CZARNECKI, J.
4802, 5034
CZECHLOWSKI, B.
4671
DABOUST, M.
3984
DAGRADI, A.E.
4382*, 4475
DALAL, N.J.
4507*
DALLEMAND, S.
5168*
D'AMICO, D.F.
4357
DANCHOT, J.
5100
DANDIA, S.D.
4686
DANI, R.
4737*, 4742*
DANIEL, E.E.
3910
DANILOV, N.V.
4762
DANILUK, J.
4802
DANKO, I.M.
4016
DANNER, D.J.
4086
DANTONO,J.
4199*
DANUS, O.
4734

DARIN, J.C.
4784*
DARLING, D.B.
4363
DAS, P.
4652*
DASAYEV, A.N.
4984
DAUCHEL, J.
3879, 3898
DAVID, H.
5103
DAVIDSON, E.
4086
DAVIDSON, G.P.
5014*
DAVIES, E.R.
4609*
DAVIES, P.L.
3789*
DAVIS, R.E.
4482*
DAWSON, A.M.
4605*
DAYTON, P.G.
4084
DE BERNARDINIS, G.
4946*
DE BRUNHOFF, M.
5031
DE CARVALHO WHITAKER, J. DE C.
4527
DE DOMBAL, F.T.
4297
DE GROOTE, J.
4285, 4931
DE LA CALLE, F.
4563
DE LA TOUR, J.
3979*, 3980*
DE MARCO, F.
3969
DE PAU, D.
5038
DE PEYER, R.
3839
DE TOGNI, F.
4139
DEAKIN, A.S.
5095
DEBAS, H.T.
3936*, 3946*, 3981*
DEBRAY, C.
3979*, 3980*, 4269, 4881
DEBUIRE, B.
4914*
DEČ, J.
4714
DECARLI, L.M.
3997*
DECHELETTE, M.A.
4155*
DECKER, H.R.
4667
DEDERER, I.U.M.
4831
DEKHKANOV, T.D.
4127
DEKUSH, P.G.
3900
DEL TACCA, M.
3863*
DELAINI, E.T.
4160
DELBARRE, F.
5100
DELEEUW, G.
4050
DELEVA, ZH.I.
3884
DELIOT, J.F.
4577
DELL ANNA, B.
4550
DELL'ANNA, B.
4406
DELMONT, J.
4407
DELOLME, H.
4416
DELPECH, A.
3803
DEMLING, L.
3887, 4248, 4258, 4403*
DEMOFONTI, A.
4828
DEMOS, N.J.
4328
DENBESTEN, L.
4301
DENNIS, R.L.
3804
DENTICO, P.
4897
DEPERREX, U.
5059
DEPIERRE, J.W.
4067
DERKSEN, D.H.
4072
DERUYTTERE, M.
4931
DESAI, H.G.
4504*
DESCOS, L.
4727
DESMET, V.J.
4285
DETERLING, R.A. JR.
4717
DEVAUX, M.A.
3865*
DEVIC, G.
4833
DEWULF, E.
4625
DEYHLE, P.
4196*, 4395, 4415, 4423, 4659, 4684
DEYHLE, P
5174*
DEYHLE, P.
5189
DHAR, G.J.
4476
DHUMEAUX, D.
4942*
DI MAGNO, E.P.
4723*
DI MARTINO, A.
4828
DI PIETRANTONIO, M.
4038, 4136
DI PIETRO, P.
4840
DI SOMONE, A.
4116, 4812
DIACHENKO, P.K.
4410
DIAZ-FLORES, L.
4892
DIAZ ISEA, A.
4811
DIAZ WATSON, C.
5065
DICK, R.
4929
DICKINSON, P.B.
4287, 4288
DIDATO, A.
4814, 4826
DIESTCHY, J.M.
3859
DIETZE, F.
4616, 4773
DIKAIA, L.
3948
DIKSHTEIN, E.A.
4367*
DIMARINO, A.J.
3905
DINTSMAN, M.
4372*
DIXON, K.
5164*
DJAHANGUIRI, B.
3929*
DLUSKA, J.
5047
DMITRIEV, A.N.
4208
DOBBINS, W.O. III
3785*
DOBRONTE, Z.
4205
DODDS, W.J.
3873*, 4299, 4817
DOLGOR, P.
4808
DOLLINGER, H.
4226
DOMINGO, N.
3975*
DOMINGUEZ, A.
4792*
DOMSCHKE, S.
3887
DOMSCHKE, W.
3897, 4157, 4403*
DONCHENKO, G.V.
4113
DONIACH, D.
4796*
DONNER, D.
4359

DONOVAN, J.F.
4593
DORF, G.
4800
DORNER, M.
3965
D'ORSI, C.J.
4199*
DOTEVALL, G.
3952
DOTTER, C.T.
4184
DOUSTE-BLAZY, L.
4741*
DOUTRE, L.P.
4959
DOWN, W.H.
4059
DRACK, E.
3825
DRAINER, I.K.
4320
DRASAR, B.S.
4169
DRASKOVICOVA, M.
5107
DREILING, D.
5177, 5179
DREILING, D.A.
4766, 4776
DREZE, C.
4468
DRIESSEN, W.M.W.
4216
DRILLET, F.
3826
DU CAILER, J.
4436
DUBERMAN, L.B.
4973
DUBOIS, A.
3874*
DUBOVA, M.N.
4211
DUBRASQUET, M.
3956, 3963
DUCA, S.
4102
DUDA, K.
4453
DUDA, P.
4874*
DUDZIAK, P.
4672
DUFFELL, D.R.
4622
DUGAL, P.
4940*
DUGAS, M.C.
3856
DUGLADZE, D.I.
4029
DUHAMEL, B.
4645*, 4653*
DUMBADZE, G.G.
4028
DUNCAN, E.H.L.
4778
DUNPHY, J. E.
4528
DUNPHY, J.E.
4650*
DUPONT, C.
3918
DUQUESNEL, C.
4727
DURAND, S.
4741*
DURHAM, J.P.
3912
DURR, H.K.
4247
DUTHIE, H.L.
4526, 4530
DUTZ, W.
5078
DUWAT, R.
3984
DVORCHIK, B.H.
3787*
DWIGHT, R.W.
4497*
DWYER, W.A.
4598
DYBAN, P.A.
4131
DYDUCH, A.
5045, 5049
DYSON, J.L.
4710*
DZAROKHOKHOV, K.I.
4111
DZHORBENADZE, T.A.
4028
EBATA, H.
4027, 4074, 4104
EBERHARDT, G.
3880
ECCHER, C.
5005
ECKHARDT, R.
5184
ECOBICHON, D.J.
4056
ECONOMOPOULOS, P.
4520
EDDLESTON, A.L.W.F.
4901*
EDELMAN, M.J.
4553
EDMONDS, C.J.
3904
EDWARDS, J.P.
4709*
EDWARDS, P.O.
4859*
EECKELS, R.
4865
EFIMOV, V.S.
4753
EGGER, G.
4191*
EICHHORN, H.J.
4355
EIGENBRODT, E.H.
4786*, 4875*
EIKMAN, E.A.
4940*
EISELT, E.
4534
EISEMAN, B.
5085
EISENBERG, D.
4545*
EITNER, K.
4843
EL ROUBI, A.
5077
ELDER, J.B.
4511*
ELGORT, D.A.
4823
ELGUEZABAI, A.
4373*
ELLIOT, W.H.
4013*
ELLIOTT, D.W.
4379*
ELLIOTT, W.H.
4128
ELLIS, F.
4360
ELLIS, J.T.
3804
ELSAS, L.J. II
4086
ELSTER, K.
4383*, 4531, 5043
EMAS, S.
3965
EMASE, S.
3956
EMTAGE, J.S.
4163
ENDRE, L.
5129
ENE, M.
4874*
ENGEL, J.J.
4862*
ENGELHARDT, A.
4437
ENGELHART, G.
5186
EPIFANOV, N.S.
4010
EPSTEIN, S.S.
3993
ERAKLIS, A.J.
4576
ERB, W.
5004
ERIKSSON, M.
5127
ERNSTER, L.
4067
ERSAY, C.
4775
ERSPAMER, V.
3962
ESAKI, T.
5101
ESENKO. I.P.
4842

ESMAILI, H.
5078
ESPINOZA, J.
4161
ESQUERRE, F.
4359
ESSA, N.
3937*
ESSIOUX, H.
4894
ESTRIN, J.
4679
EVANS D.C.
3933*
EVANS, D.J.
4629
EVANS, W.H.
3794*
EWE, K.
3830, 3841
EWERS, H.R.
3955
EWING, M.R.
4729
EYMARD, J.
4833
FABIANOWSKI, J.
4906*
FAEGENBURG, D.
5168*
FAGIOLO, U.
4903*
FAHRLANDER, H.
4424
FAIN, J.N.
4080
FAITELBERG, R.D.
3827
FAKTOR, V.M.
4144
FALKENHAGEN, D.
4787*
FANI, C.
4991
FARA, J.W.
3872*
FARBAKYOVA, G.
5107
FARBEROVA, I.Z.
4031
FAREDIN, I.
4849
FARELLO, G.A.
4139, 4357, 4818
FARMAN, J.
5168*
FARMER, R.G.
4947*, 5171*
FARZANEH, I.
5112
FASSKE, F.
4104
FASTH, S.
3864*
FATEYEVA, V.I.
3914
FAUST, H.
4332
FAVIA, G.
4357
FAVRE, R.
4994
FAXELIUS, G.
4854*
FEDEROV, V.D.
4238
FEDUROV, V.V.
4090
FEHR, H.F.
5188
FEICHTER, G.E.
4463
FEIGELSON, J.
4920*
FEINENDEGEN, L.E.
4735
FEIST, D.
4839
FEKLISOVA, L.F.
5032
FENDER, H.R.
5093
FENTON, J.C.B.
4654*
FERA, G.
4897
FERGUSON, A.
4607*
FERGUSON, R.
5162*
FERNANDES, J.
5106
FERRANTE, W.A.
4340
FERRAZ RAMOS, J.
4206
FEURLE, G.
4699
FEUSTEL, H.
4667
FIANCHINTA, A.
4895
FIASSE, R.
4938
FIEDLER, A.
4098
FIELD, M.
4152*
FIGUEROA, C.
4734
FIHEY, M.A.
4564
FILLER, D.
4667
FILLIPPINI, L.
4417
FINLAYSON, N.D.C.
5126
FISHER, R.S.
3905
FISHZON-RYSS, I.U.I.
4820
FLEGEL, U.
4190*
FLEISCHER, K.
3983, 4772
FLEMING, K.
4148
FLEMSTROM, G.
3842, 3843
FLENDRIG, J.A.
4216
FOLLY, G.
4166*
FOMIN, V.B.
4448
FONTAINE, J.L.
4606*
FONTANIN, O.
4139, 5005
FORDTRAN, J.S.
4488*
FORDTRAN, , J.S.
4491*
FORELL, M.M.
3988, 4247
FORNES, M.F.
4295
FORREST, J.A.H.
5126
FORTIS, P.A.
4725
FORTNER, J.G.
4819
FOSTER, J.H.
4834
FOX, H.
5166*
FRAGOAS, M.
4567
FRANCO, D.
4942*
FRAYSSINET, C.
4034
FREDGA, A.
4071
FREITAG, H.
4383*
FREMLIN, J.H.
4866
FRENCH, S.W.
3789*
FREY, C.F.
4790*
FREYBERGER, H.
5057
FREZZA, M.
4117
FRIEDMAN, M.A.
4081
FRIESEN, H.G.
4099
FRITSCH, W.P.
3961, 4398, 4399
FRITZ-MIKULSAK, V.
4336
FRIZZELL, R.A.
3856
FROLKIS, A.V.
4493*
FROMM, H.
4617, 5041
FROMMHOLD, H.
4277

FROMMHOLD, W.
4273, 4315
FROT-COUTAZ, J.
4093
FRUCHART, J.C.
4914*
FRUHAUF, S.
3988
FRUHMORGEN P.
5185
FRY , L.
4639
FUCHS, K.
4759
FUCIK, M.
4534
FUJIWARA, K.
5105
FUJIWARA, Y.
3796
FUKUYAMA, T.
4252
FULOP, T.
4910
FULTON, R.E.
5022
FUMAGALLI, I.
4997
FUNK, C.
4425, 4997
FURBERG, B.
4378*
FURMANENKO, E.D.
4267
FURTADO, M.
4742*
GABBE, E.E.
5091
GABRYLELEWICZ,A.
4757
GACS, M.
5030
GAFFI, A.
5038
GAGUSHIN, V.A.
4989
GAIDICHUK, S.T.
4512*
GAINES, S.A.
4183
GAJDOS, A.
4049
GALANTI, B
4879
GALASINSKA-POMYKOL, I.
4596
GALASSO, U.
4724
GALBRAITH, R.M.
4901*
GALL, D.G.
5020
GALLEY, C.B.
4180
GALLI, E.
4840
GALLO, G.A.
4948*
GALLO, L.
3974*
GALPERIN, E.I.
4990
GALZAYD, E.
4361
GALZIGNA, L.
4121
GAMA-RODRIGUES, J.J.
4651*
GAMMARROTA, V.
4867
GAMULIN S.
4088
GANDOLFI, P.
4139
GANELINA, L.S.
4142
GANGOLLI, S.D.
3818*, 3917
GANGRADE, R.R.
4507*
GANS, S.L.
4291
GARABIOL, B.
5145
GARCIA BENGOECHEA, M.
4830
GARCIA, H.
4015*
GARDE, J.
4833
GARDNER, J.D.
3817*
GARON, G.
5132*
GARRETT, J.R.
5125
GARWICZ, S.
4556
GASSMAN, R.
4458
GASSMANN, R.
4426
GATMAITAN, Z.
4181
GATOS, M.
4669
GAUGUA, A.M.
4028
GAUTIER-BENOIT, C.
3984
GAUTIER-BENOIT, G.
4592
GAUTIER, C.
4094
GAY, T.
4780*
GAZE, H.
4656*
GEBEL, B.
4462
GEBHARD, R.L.
5159*
GEENEN, J.E.
4666
GELAIN, J.
4833
GELFAND, D.
4204
GENDRON, Y.
4894
GENTILI, B.
4828
GENTILINI, M.
5145
GENTILINI, P.
4856
GENYK, S.N.
4770
GEORGE, P.
4188*
GEORGOULIS, J
4669
GERARD, A.
4965, 4993
GERASIMOV, A.M.
4076
GERHARD, H.
4060
GERLACH, U.
4262
GERO, W.
4265
GERONILLA, D.R.
5015*
GEUBAL, A.
4900*
GHAZY, F.S.
3834
GHESQUIER, D.
3947
GHISOLFI, J.
4470
GIANETTA, E.
4039
GIBSON, J.A.
5083
GIBSON, J.J.
4654*
GICHON, G.
4186*
GIGNOUX, M.
5054
GIGON, P.L.
4138
GILCHRIST, J.
4188*
GILL, C.C.
5173*
GILLESPIE, G.
5178
GILLESPIE, W.J.
4744*
GILLET, B.
5070
GIN, A.C.
3869*
GIOVANINETTI, G.D.
4160
GIRARD, M.
4392, 4670
GIRMA, J.P.
3963
GIRMANN, G.
4848

GIRODET, J.
3947
GISLON, J.
3963
GLASEL, E.
4584
GLIDZIC, V.
4683
GLOOR, F.
5186
GLUSHKOV, I.U.A.
4326
GMAZ-NIKULIN, E.
4108
GMELIN, K.
4839
GNANDIGER, H.P.
4463
GNAUCK, G.
4844
GO, V.L.M.
4723*
GO, V.W.L.
3986, 4736
GOBEL, D.
4221, 4626
GODQUIN, R.
4994
GOEBELL, H.
3953, 3954, 4494*, 4767
GOGIBERIDZE, G.V.
4107
GOHRBAND, G.
4435
GOLAND, L.G.
3987
GOLDBERG, H.I.
4817
GOLDMAN, M.
4184
GOLDSTEIN, N.P.
4782*
GOLIGHER, J.C.
4508*, 4525, 4709*
GOLLER, I.
5014*
GOLOVINA, N.E.
4231
GOLUBOV, I.S.
4799
GOMEZ CONDE, S.
5042
GOMEZ, J.A.
3805
GOMEZ-PAN, A.
3944*
GONCI, L.
5008
GOODHART, J.M.
5170*
GORBUNOV, V.V.
5012
GORDON, M.J.
3938*
GORIUKHINA, T.A.
4173
GORSHKOV, V.A.
5027
GORSKA-DUBOWIK, M.
4671
GOSCHKE, H.
4581
GOSSELIN, B.
4366*
GOT, R.
4007*, 4093
GOTTESBUREN, H.
3815*
GOTZE, H.
4934
GOUFFRANT, J.M.
4959
GOUGH, A.L.
4519
GOUNOT, J.
4416
GOURLEY, B.
4540*
GOYAL, R.K.
4333
GRACEY, M.
5088
GRAHAM, D.Y.
4333
GRAHAME-SMITH, D.G.
5120
GRAINGER, J.
4441
GRANDJEAN, C.
4015*
GRANT, A.G.
4126
GRANT, B.
3958
GRANT, B.J.
3942*
GRASSI C.
4010*
GRASSI, G.G.
4010*
GRASSI, P.E.
4867
GRASSO, P.
3917
GRATZL, M.
4848
GREEN, F.H.Y.
5166*
GREENSTEIN, A.J.
4776, 5163*, 5177, 5179
GREEP, R.O.
3804
GRELAC, F.
3947
GRENIER, J.F.
3879, 3898
GRESHILO, M.S.
5072
GRIFFEN, JR.
4219
GRIFFEN, W., JR.
4244
GRODSINSKY, C.
4953*
GROISMAN, S.D.
3900
GROMOVA, O.
3999*
GROSSMAN, M.I.
3936*, 3946*, 3981*, 4471
GROTELUSCHEN, B.
4522
GROTH, U.
4618
GRUMMER, R.H.
3920*
GRUN, M.
4115
GRUND, E.R.
3973
GRUNDMANN, E.
4457
GRUNDY, R.K.
4059
GUELLER, R.
4472
GUELZOW, M.
4787*
GUERRERO, J.A.
4600
GUERRIN, F.
4366*
GUEVIN, R.M.
4893
GUISANTES, J.A.
4193*
GUISTI, G.
4879
GUIVARCH, M.
4747
GUKASIAN, A.A.
4445
GULESSERIAN, H.P.
4788*
GULI, E.P.
4687
GULY, M.F.
3959, 4022
GUNTHER, R.
4532
GUPTA, C.K.
4652*
GUPTA, D.N.
4375*
GUPTA, M.M.
4904*
GUPTA, N.M.
4705
GUPTA, S.P.
4380*
GUREVA, T.A.
5032
GUREVICH, G.T.
5076
GUSEV, AI.
4823
GUSEVA, N.G.
4338
GUSTAFSON, A.
4955*
GUTH, P.H.
3939*
GYURKOVITS, K.
5129

HABERKORN, S.
4651*
HARR-GAMA, A.
4651*
HARTE, D.
5016*
HASHIYA, T.
4223, 4228, 4252
HADDAD, J.K.
4300, 4331, 4472
HADORN, B.
4540*
HAGEMANN, E.
4933
HAGER-MALECKA, B.
5049
HAGGITT, R.
4360
HAHN, R.G.
4657*
HAINES, D.S.M.
4072
HALKIN, A.P.
4063
HALL, R.
3944*
HALLADAY, S.C.
4057
HALLER, C.V.
4032
HALSBAND, H.
4938
HALSTED, C.H.
4623
HALTER, F.
5187
HAMADA, N.
4223, 4228, 4233, 4252
HAMILTON, J.R.
5020
HAMLY, C.
5018*
HAMMER, B.
5186
HANAK, H.
4821
HANITEVYCH, Y.V.
4130
HANSBURY, E.
4043
HANSEN, D.H.
4412
HANSEN, T.
3949
HANTSCHMANN, N.
4571, 4678
HARDACRE, J.M.
4949*
HARDING, R.
3919
HARDY, C.C.
3911
HARDY, E.
3984
HARING, R.
4101
HARNED, R.K.
4431
HARRIGAN, D.J.
3858
HARRINGTON, G.
4784*
HARRIS, D.
3922*
HARRIS, J.B.
3930*
HARRISON, G.A.
3804
HART, M.C.
5112
HARTROFT, W.S.
3804
HARTWICH, G.
4157
HARVEY, R.F.
4609*
HARVEY, T.C.
4866
HASHIKAWA, T.
3881
HASHIMOTO, Y.
4192*
HASUMI, K.
3993
HATA, Y.
4645*, 4653*
HATCH, E.I.
4829
HATIYA, T.
4233
HATTORI, K.
4289
HAUGER, W.
4240
HAUPT, H.
4074
HAUSAMEN, T.U.
3961, 4398, 4399
HAUTON, J.
3975*
HAVEN, G.T.
4055
HAVIA, T.
4353
HAWK, W.A.
5171*
HAWORTH, J.C.
4540*
HAYAMA, J.
3968
HAYANO, I.
4459, 4682
HAYASHI, A.
3881
HAYASHIDA, S.
5092
HAYLES, A.B.
5111
HAYMAKER, W.
3804
HEADING, C.E.
3818*
HEANEY, R.P.
3845
HEBERT, G.
5109
HECHTMAN, H.B.
4497*
HECKER, W.C.
4334
HEFTI, M.L.
4426
HEILMANN, K.
4413
HEIM, M.
4443
HEIMLICH, H.J.
4343
HEINE, W.
3828
HEINISCH, M.
5184
HEINRICH, H.C.
5091
HEINZ, F.
4149*
HEISKELL, C.A.
4675
HELLWEGE, H.H.
4876*
HELPAP, B.
4408
HENDERSON, R.
5088
HENDRY, J.H.
4179
HENRICH, M.
4995
HENRY, D.P.
3874*
HERMAN, D.L.
3851
HERMANIUK, I.A.L.
4087, 4089
HERMS, G.
4624, 4882
HERNANDEZ-ALMENAS, N.
5150
HERNANDEZ GUIO, C.
4792*
HERRERIAS, J.M.
4264
HERRING, J.
3911
HERZIG, M.
4451
HESCHI, R.
5001
HESS, C.
5058
HEYMANN, H.
5035
HIENZ, H.A.
4350
HIGGS, R.H.
4486*
HIGUCHI, Y.
4411
HILL, G.L.
4709*
HILL, M.
4307
HILLYER, G.V.
5150

HIND, C.R.K.
3911
HIRNER, A
4101
HIROTA, T.
4387*
HIRSCHOWITZ, B.I.
4509*
HIRSH, F.S.
5128
HO, L.
4691
HODGSON, J.
5122
HOEKSTRA, W.G.
3920*
HOFFBRAND, A.V.
4634, 4639
HOFMANN, A.F.
4941*
HOGAN, W.J.
4299
HOGAN W.J.
4666
HOHN, P.
4825
HOLCOMB, G.W.
4834
HOLCROFT, J.W.
4650*
HOLDEN, R.J.
4607*
HOLDSWORTH, G.
4009*
HOLKER, B.
3841
HOLLENBECK, J.I.
4001*
HOLLENDER, L.F.
4582, 4751, 4768
HOLMES, G.K.T.
4636
HOLMES, I.H.
5014*
HOLSTEN, D.R.
4276
HOLTERMULLER, K.H.
3986, 4736
HOLTON, C.P.
4829
HOLYOKE, E.D.
4646*
HOLZEL, W.
4280
HOMMEL, G.
4745*
HONDA, R.
4662
HONG, S.K.
3862*
HONGO, M.
5092
HONGQUE, M.P.
4625
HONIGMANN, G.
4844
HOPF, U.
4880
HOPNER, F.
4334
HORACEK, F.
4322
HORAK, W.
5002
HORMAN, D.
4324
HORROCKS, J.C.
4297
HOSOYA, N.
4162
HOTZ, J.
3953, 3954, 4494*, 4767
HOUCKE, M.
4592
HOUSE, A.K.
4690
HOUSE, R.K.
4235
HOUTHOOFT, D.H.
4572
HOVUMPA, A.
4793*
HOVZE, Y.H.
4063
HOWARD, J.M.
3906
HOWAT, J.M.T.
4951*
HOWES, R.
4739*
HOWLETT, P.J.
4526
HRITZ, A.
4161
HRYTSENKO, N.M.
3832
HUBNER, R.
4933
HUCAL, G.
5151
HUETTER, J.
4787*
HUGEL, H.
5186
HUITRIC, E.
3803
HULL, A.R.
4786*
HULSMANN, W.C.
3849
HULTEN, L.
3864*
HUMASON, G.L.
3804
HUORANSZKI, F.
4351
HUPPERTZ, A.
4898
HURWITZ, A.
4331
HURWITZ, A.L.
4300
HUSMANN, K.H.
4276
HUSSON, A.
4094
HUSSON, J.M.
4881
HUTHWOL, B.
3888
HUTTER, J.
5084
HWANG, S.W.
3998*
IAKHONTOVA, O.I.
5099
IAKOVELEA, I.A.
4422
IANCU, A.T.H.
4613
IANCU, A.V.
4613
IAREMKO, E.O.
4158
IAZOVA, A.K.
4823
IDA, K.
4192*
IGODT-AMEYE, L.
4865
IHSE, I.
4738*
II, K.
4358
ILSON, R.G.
4009*
IMBEAU, S.A.
4473
IMBESI, C.
4573
IMPETA, D.
3962
INAKOSHI, H.
4411
INBERG, M.V.
4353
INDRAYAN, A.
4652*
INGIS, D.A.
4947*
INGRAND, J.
5100
INOUE, K.
4233
IQBAL, Z.M.
3993
IRGASHEV, S.B.
3801
ISANIN, N.A.
4146
ISELER, G.
4020
ISHCHUK, O.E.
4267
ISHIDATE, T.
3796
ISMAN, H.
4033
ISOMAA, B.
3823
ISRAILI, Z.H.
4084
ISSELBACHER, M.J.
4871*

ISSHIKI, S.
4662
ITO, S.
4459
ITOH, K.
5105
IUDIN, L.A.
4911
IUDKOVSKAIA, I.L.
4224
IUSCHUCK, N.D.
5098
IVANNIKOV, N.F.
4987
IVEY, K.J.
3935*, 4396
IWADARE, J.
4662
IWAMURA, K.
4230
IZANT, R.J. JR.
5104
JABLONSKA, M.
4534
JACOBS, G.
5007
JACOBSON, E.D.
3925*
JAECK, D.
3879, 3898
JAEGER, E.
3887
JAHADI, M.R.
4649*
JAHNBERG, T.
3864*
JAILLARD, J.
4914*
JAKAB, F.
5008
JAMES, A.E. JR.
4195*
JAMES, I.
4805
JAMIESON, J.C.
4085
JAMIESON, J.D.
3976*
JANSON, K.L.
4568
JARNEROT, G.
5160*, 5161*, 5165*
JASO, E.
5082
JAVORS, B.
4696
JEAN, E.
3918
JEFFERSON, L.S.
3811*
JELENKOVIC, M.
5139
JENNEWEIN, H.M.
4403
JENNINGS-KHODADAD, E.
5078
JERKSY, J.
4004*
JEZEQUEL, Y.
4894
JHA, B.N.
5154
JODL, J.
4612*
JOHANSEN, K.S.
4924*
JOHANSEN, T.S.
4924*
JOHANSSON, H.
3943*, 4378*
JOHNSON, L.F.
3862*
JOHNSON, L.R.
3868*
JOHNSON, P.
4955*
JOHNSON, W.C.
4497*
JOHNSTON, D.
4508*, 4524, 4525, 4529
JOHNSTON, R.E.
4058
JOLLER-JEMELKA, H.
4934
JONES, C.B.
4951*
JORDAN, J.J.
4019
JORNET, J.
4600
JOS, J.
3826
JOSEFSSON, K.
5119
JOSEPH, T.P.
4469
JOUBAUD, F.
4302
JOVANOVIC, B.
4483
JULESZ, J.
4849
JULIEN, R.
3975*
JUSKIEWENSKI, S.
4470
JUSZCZYK, J.
4908
JUVARA, I.
4961
KACZMARSKI, M.
4883
KAESS, H.
3965, 5059
KAGAN, I.G.
4282
KAHN, P.
4821
KAIFU, S.
4459
KAISER, D.
3825, 4934
KAISER, E.
4910
KAISER, G.
4745*
KAKIZAKI, G.
3796
KAKUMU, S.
4837
KALHE, I.
4616
KALIMA, T.V.
4369*
KALIMAN, P.A.
4017
KALINOVSKI, V.P.
4173
KALKAY, M.N.
4329
KALOS, A.
4664*
KAMINSKI, D.L.
3977*
KAMISAKA, K.
4181
KANETI, J.
4599
KANIAK, J.
4899*
KAPLAN, R.
4161
KAPOOR, C.L.
4122
KAPP, F.
4332, 4424
KAPP, S.
4232, 4618
KARBACH, Y.I.
4130
KARCZEWSKA, K.
5045, 5049
KARHADE, N.V.
4908*
KARIAKINA, I.A.
4076
KARIN, S.N.M.
3961
KARK, A.E.
5168*, 5177, 5179
KAROLCEE, J.
5107
KARTASHOVA, O.I.A.
4799
KARYDIDI, P.G.
4762
KASAOKA, C.
4489
KASPER, H.
4246
KASPEREK, K.
4739
KASSER A.
4894
KASTIN, A.J.
4464
KASUGAI, T.
4971*
KASZEWSKA-JABLONSKA, I.
4671
KATOCH, Y.
5101
KATONA, Z.
5129

KATSUKI, T.
4057
KAUFMANN, M.
5059
KAUFMANN, P.
5060
KAWACHI, T.
4387*
KAWADA, T.
4057
KAWAI, K.
4192*, 4229, 4487*
KAWARITA, K
4864
KAWARADA, R.
4864
KAWARDA, R.
4863
KAWARDA, Y.
4385*
KAZIL, P.
4322
KAZNACHEEV, V.S.
4045
KEARLEY, R.W.
3868*
KEDA, B.I.
4755
KEDDIE, N.C.
4519
KEHRLI, P.
4934
KEIGHLEY, M.R.B.
4484*
KEKONAKI, M.
4480
KELKAR, S.S.
4902*
KELLER, N.
5187
KELLY, M.
3866*
KELLY, P.A.
4099
KELNER, R.T.
4860*
KEMENY, P.
4321
KENNEDY, T.
3866*
KERDZAIA, A.O.
4028
KERR, J.C.
4037
KERSTEIN, M.D.
5138
KESTENS, P.J.
4938
KETS, E.
4551
KETTLER, L.H.
4844
KHACHADURIAN, A.K.
5018*
KHANA, M.N.
5154
KHANNA, S.K.
4908
KHATTAB, A.K.
5087
KHINTS, R.
4042
KHOKHLOV, A.P.
4107
KHOMIAKOV, A.E.
4755
KHOMINSKAIA, Z.B.
4109
KHOMUTOVSKY, O.A.
4112
KHONELIDZE, G.B.
4422
KHOREV, G.N.
4748
KHOURY, P.
4569
KHUTIEV, T.S.S.
4809
KIA, D.
4187*
KIDD, E.G.
5170*
KIEFER, G.
4053
KIERSKA, D.
4159
KILAM W.K.
4707
KILPATRICK, Z.M.
5131*
KIM, D. N.
4129
KIM, U.
5180
KIM, Y.
4065
KIM Y.S.
4065
KIMURA, S.
4194*
KIMURA, T.
3834
KINALSKA, I.
4757
KING, M.
4052
KINGSLEY, D.
4441
KINOSHITA, Y.
4352
KIRSCHNER, P.
4825
KIRSNER, J.B.
4289, 4658
KITAZAWA, S.
3860
KITTLICK, M.
4171
KIUCHI, Y.
5105
KIVILAAKSO, E.
4369*
KIYOHARA, A.
4391*
KLAEVEMAN, H.L.
3817*
KLAEVMAN, H.L.
5159*
KLEIN, G.
3804
KLEIN,V.E.
4214
KLEINTEICH, B.
4546, 4584
KLEYBRINK, H.
3961, 4398, 4399
KLIMENKO, A.I.
4042
KLIMINSKII, I.V.
4492*
KLINE, T.S.
4236
KLIPSTEIN, F.A.
4604*
KLOTZ, V.
4793*
KNAUER, C.M.
4307
KNOBEL, S.
4178
KNOBLAUCH, M.
4425, 4779
KNOLL, J.
3890
KNORR, D.
3846
KOBAYASHI, S.
4371*
KOBIERSKA, A.
5048
KOCH, A.
4403
KOCH, C.W.
4213
KOCH, H.
4171, 4248
KOCIAN, J.
4619
KOCIANOVA, J.
4552, 5051
KOELSCH, K.A.
4325
KOGEL, H.
4668
KOGURE, K.
4387*
KOHLER, J.
5041
KOHLI, Y.
4229
KOHLI,Y.
4487*
KOHOUR, E.
5078
KOHUT, A.
4538
KOISCHWITZ, D.
4218, 4255, 5175
KOJECKY, Z.
4591
KOKUE, E.
3968
KOLACZEK, F.
4383*

KOLLIN, J.
4266
KOLODEJ, A.
3861*
KOLOMIYSTEVA, I.K.
4045
KOLUPEAVA, G.N.
4270
KOMARKOVA, A.
4263
KOMAROVSKY, V.S.
4448
KOMAROWSKA, M.
4505*
KOMI, N.
4358
KOMURO, T.
3860
KONDAKOVA, A.E.
4110
KONDE, W.N.
4133
KONDO, H.
4223, 4252
KONDO, I.
4863, 4864
KONDO, T.
4459
KONTUREK, S.
3978*
KONTUREK, S.J.
3931*, 3981*
KOPELEVICH, V.M.
4075
KOPIN, I.J.
3874*
KOPAJKIC, M.
5139
KOPP, G.
4898
KOPPARLAG, C.
4880
KORENEV, N.N.
4979
KOREPANOV, V.I.
4270, 4810
KORMANO, M.
4164*
KORNYUSHYNA, T.V.
4063
KOROLENKO, T.A.
4110
KOROLEV, B.A.
4989
KOROLEVA, N.P.
4432
KOROSTOVTSEVA, N.V.
4145
KOROTKORUCKO, V.P.
4016
KOSAKA, K.
4014*, 4061
KOSAKA, Y.
4863, 4864
KOSHARKO, K.A.
3838
KOSHEVSKAYA, L.I.
4974
KOSINSKI, J.
5034
KOSS, N.
5138
KOSSAKOWSKI, R.
4870*
KOSTER, J.F.
5106
KOSTERLITZ, H.W.
3891
KOSYAK, E.P.
4594
KOTELES, G.
4321
KOTSCHY, M.
4899*
KOTWAL, S.E.
4902*
KOUBA, R.
4972
KOVALCHUK, V.K.
3793*
KOVALENKO, V.F.
3832
KOVALENKO, V.M.
4113
KOVTUN, T.D.
4130
KOWAL-GIERCZAK, B.
4899*
KOWALCZYK, J.
4453
KOWALCZYK, L.
4930
KOWALCZYK, T.
3920*
KOWALEWSKI, K.
3861*
KOWLESSAR, O.D.
4764
KOYAMA, Y.
4387*
KOZAK, J.
4802
KOZEK, W.J.
5157
KOZUKA, S.
4837
KRAFT, G.
4413
KRAFT, L.M.
3804
KRAJEWSKA, B.
5048
KRAJKA, K.
4967
KRAKOVIC, M.
4674
KRAKOVSKY, A.I.
4987
KRAMER, G.
4624, 4882
KRAMER, L.
3855
KRAMHOFT, J.
4967
KRANTZ, B.B.
4795*
KRAUS, L.
4478
KRAUSE, P.H.
4149*
KRAUSE, U.
3943*, 4378*
KRAUTHEIM, J.
5066
KRAVCHENIO, L.V.
4041
KRAVCHENKO, L.V.
3797
KRAVCHUK, I.P.
4241
KREEL, I.
5180
KREEL, L.
4220
KREJOVIC, B.
4347
KREJS, G.
4423
KREJS, G.J.
3889, 4339, 4515
KREMENCHUZKY, S.
4926
KREMER, B.J.
4763
KRENTZ, K.
4435
KREUTZER, W.
4053
KREUZER, J.
4314
KRIEGEL, H.
4438
KRINITSKI, S.S.
4135
KRISHNAMACHARI, K.A.V.R.
4861*
KRIVOSHEENKO, T.N.
4145
KRIVULIS, D.B.
4140
KRIVULIS, D.B.
4080
KROKHINA, E.I.
4175
KRONBORG, O.
4510*, 4987
KRTSCH, H.
4308
KRUTSKIKH, E.V.
5009
KRYLOVA, N.P.
4831
KRYLOVA, T.V.
4975
KRYSL, I.
4281
KRYSTEV, L.P.
3797, 4041
KRZEMIEN
4055
KUCERA, J.
4660
KUCEROVA, I.
4888

KUCHARCZYK, B.
3824
KUCZYNSKA-SEKIETA, K.
4714
KUDRMANN, J.
4552
KUDRYAVTSEV, B.N.
4079
KUDRYAVTSEVA, M.V.
4079
KUGELBERG, C.
4738*
KUTTUNEN, P.
4480, 4603*
KUKUSHKIN, G.S.
4029
KULAKOWSKI, S.
4848
KULENKAMPFF, H.
3800
KULHARNI, B.S.
4499*
KUMAR, P.
4652*
KUMAR, P.J.
4605*
KUNATH, U.
4314
KUNNEN, M.
4869
KUNTZMAN, R.
4083
KURITA, G.
4460
KURPAKOV, N.A.
5136
KUSHA, I.
4520
KUTZ, K.
4191*
KUZINA, M.F.
5076
KUZNETSOV, V.P.
5032
KUZNETSOVA, G.B.
4146
KVITSINSKAIA, E.A.
4035
KVITSINSKAYA, E.A.
4140
KYALWAZI, S.K.
4794*
KYRIAKOU, K.
4168*
LA SALA, M.A.
5191
LABARRAQUE, J.P.
4645*
LABAS, R.
4746
LABOW, S.B.
4689
LACHECKI, Z.
5146
LADEN, G.E.
4654*
LADOUCEUR, J.
5109
LAFONT, H.
3975*
LAI, E.
4052
LAINEE, J.
4564
LAINEE, R.
4564
LAIRD, T.
3804
LAIRON, D.
3975*
LAKE, B.G.
3818*
LAKTAEVA, T.V.
3989
LAM, A.A.
4040
LAMBERT, R.
4155*, 4513
LAMERS, J.M.J.
3849
LAMMLI, J.
4425
LANAS LARRANAGA, E.
4830
LANCASTER, C.
4501*
LANDBERG, T.
4556
LANDOR, J.H.
4479
LANDRIEU, P.
4785*
LANE, A.E.
4605*
LANGAZO, Z.P.
3960
LANGE, H.
4250
LANGE, H.H.
4698
LANGER, S.
4408
LANGERON, P.
4966
LANKISCH, P.G.
4759, 5026
LANSDOWN, A.B.G.
3792*
LANSKAYA, A.B.
3802
LANZA, F.
4381*
LAPIRRE, J.
5144
LAPP, M.
4429
LARGIADER, F.
4779
LARKIN, E.
4554
LARRAIN, F.
4734
LARSEN, J.K.
4412
LARSON, S.M.
4195*
LASTIC-MALETIC, S.
5139
LASZLO, P.
4351
LATASTE, J.
4421
LATIMER, R.G.
5155
LAUDANO, O.M.
3957
LAUDENBACH, P.
5063
LAUE, R.
4616
LAUGUTIN, V.D.
5148
LAUMONIER, R.
3803
LAUTERBACH, F.
3822
LAVERDANT, C.
4894, 5142
LAW, C.
5151
LAWRENCE, G.
4441
LAWSON, D.E.
4163
LAWSON, R.A.M.
4342
LAWSON, T.L.
3805, 4817
LAYRISSE, M.
3806*
LAZAREV, I.M.
4685
LE PICARD, P.
4421
LEACH, S.C.
3804
LEAL, S.
4742*
LEBEDEVA, N.A.
4852
LEBEL, E.
4374*
LEBENTHAL, E.
4544*
LEDNEY, G.D.
4180
LEE, E.R.
4382*
LEE, H.K.
4186*
LEE, K.T.
4129
LEE, W.M.
4901*
LEEMANN, A.
4997
LEERHOY, J.
4924*
LEETS, I.
3806*
LEFEBVRE, R.
4937
LEGER, L.
4402

LEGHISSA, G.
3995*
LEHMANN, H.
4665
LEHY, T.
3964
LEINWEBER, B.
4589
LEISING, H.
3815*
LEMPINEN, M.
4369*
LERNER, A.
5015*
LESCH, P.
4274, 4743*
LESCHINSKY, L.A.
4816
LESH, F.A.
5156
LESKO, A.
4746
LESSEL, A.
4355
LESSER, P.B.
4197*
LEUSCHNER, U.
4125
LEUTENEGGER, A.F.
4581
LEUTHOLD, E.
4426, 4458
LEVI, A.J.
4913*
LEVIN, B.
4289
LEVIN, W.
4083
LEVINA,D.M.
4823
LEVINE, W.G.
3854
LEVITSKII, A.P.
3992
LEVRAT, M.
4727
LEVY, A.G.
3817*
LEVY, G.
3858
LEWICKI, A.
4187*
LEWIN, M.
3798, 3947
LEWIS, E.P.
4650*
LEWIS, J.D.
4784*
LEWIS, J.W. JR.
5138
LEWIS, M.K.
4043
LEYMARIOS, J.
4881
LI, T.K.
4012*
LI VOLSI, V.A.
4541*
LIAW, Y.F.
4815
LIDSKY, A.T.
4982
LIE, T.S.
4027, 4074, 4104
LIEBER, C.S.
3812*, 3997*, 4154*
LIEBERTHAL, M.M.
5108
LIEBMAN, W.M.
4291
LIEHR, H.
4115
LIFSHITZ, F.
5191
LIGHTNER, D.A.
4174
LIGUORI, L.
4562
LIKHACHOV, V.A.
4756
LILLEHI, R.C.
4740*
LILLY, J.O.
5123
LIN, G.J.
4133
LIN, T.M.
3933*
LIN, W.S.J.
4815
LIN, Y.C.
3862*
LINBERG, A.
5119
LINCHENKO, I.F.
4731
LINDENBAUM, J.
3812*
LINDER, E.
4684
LINDHOLMER, B.
4711*
LINEVSKY, Y.V.
5053
LIPKIN, M.
4688
LIPPENS, R.J.J.
4681
LIPPERT, H.
3985
LIPPMAN, W.
3927*
LISCHKE, J.H.
5137
LISE, M.
3971
LISIYENKO, V.M.
4982
LISKOVA, M.
4726
LISTOWSKY, T.
4181
LITONYAN, Z.M.
4077
LIVISHITS, E.G.
4245
LIVOLSI, V.A.
4364
LLOYD, A.G.
3818*
LLOYD, J.K.
4543*
LO GERFO, F. W.
4680
LOBSIGER, M.
4841
LOBSIGER, M.M.
4339
LOCKHART-MUMMERY, H.E.
5192
LOEWE, K.R.
4588
LOFFLER, A.
4255, 4257, 4735
LOHRENZ, F.N.
4949*
LOJACONO, L.
4936
LOJDA, Z.
4612*, 5051
LOKICH, J.
4199*
LOKSHIN, L.S.
4111
LOMACHENKOV, V.D.
4428
LOMBARD, R.
4468
LOMONOSOVA, G.A.
3801
LONDONG, W.
3988
LONG, D.L.
4786*
LONG, R.J.
3852
LONGHINO, C.
4725
LONGSTRETH, G.F.
4641
LOPEZ SANCHEZ, A.
4264
LOPUKHIN, Y.M.
4029
LORENZ, G.
4850
LORENZ-MEYER, H.
3815*, 4429, 4628
LORMAN, T.B.
4430
LOSEVA, A.G.
4614
LOSITO, R.
4008*
LOUBIERE, M.
4407
LOUVEL, A.
4402
LOUVET, J.P.
5067
LOW-BEER, T.S.
4609*
LOWENFELS, A.B.
5094

LUBY, J.P.
4786*
LUCA, V.
4874*
LUDWIG, J.C.
4057
LUDWIG, R.
4443
LUIS, PALOMOA, J.
4409
LUKASOVA, A.
4322
LUKONSKA, K.
4848
LUMENG, L.
4012*
LUNA A.
4563
LUND, P.K.
3973
LUNDELL, L.
3928*
LUNDQVIST, G.
3943*, 4378*
LUNZER, M.
4807
LUPANOVA, R.I.
4986
LUPDEN, P.J.
4915
LUSCHNITZ, E.
4250
LUSHBAUGH, C.C.
3804
LUSKA, G.
4274, 4743*
LUSSIER, A.
4374*
LUTETE-KELANI, M.
4495*
LUX, G.
4203, 5043
LYAKHOVICH, V.
3999*
LYDON, S.B.
3873*, 4299
LYFORD, C.
4701
LYMBEROPOULOS, S.
4408
LYNCH, W.A.
4677
LYSAK, Z.A.
4410
MAASSEN, G.
4992
MACARTHUR, E.
4601
MACDONALD, T.T.
4607*
MACGREGOR, A.M.C.
4583
MACHAVARIANI, M.I.
4028
MACHNIK, G.
4843
MACKENSEN, S.
4214
MACKENZIE, J.F.
4376*
MACMAHON, R.A.
5021
MADAR, J.
5030
MADSEN, P.
4510*
MADYAR, Z.
4932
MAGALHAES, A.D.E.A.
4527
MAGISTRETTI, M.J.
4070
MAGNIN, G.
4024
MAGNONI, E.
4549, 4585
MAHDI, S.
3828
MAHER, J.W.
4001*
MAHIEU, P.
4938
MAIER, K.P.
4265
MAILLET, P.
4580
MAINOYA, J.R.
3808*, 3816*
MAINTZ, J.
4611*
MAIO, T.
4598
MAIR, W.S.J.
4709*
MAIWALD, L.
4418
MAKARENKO, T.P.
4809
MAKSIMOVA, V.G.
4215
MALAGELADA, J.R.
3986, 4723*
MALAGELADA, J.R.
4736
MALIK, G.B.
4375*
MALINOVSKY, N.N.
4984
MALINSKYM J.
4660
MALIUGIN, E.F.
4107
MALKOVSKA, K.
4534
MALLINSON, C.N.
5176
MALOBABIC, Z.S.
3897
MALOLEPSZY, J.
4714
MALTSEVA, I.V.
5033
MALYSHENKO, L.V.
4824
MAN, B.
4978
MANCHEV, I.
4570
MANEGOLD, B.C.
4760
MANGOLD, G.
4825
MANIFACIO, G.
4740*
MANITZ, G.
4222
MANSBACH, C.M. II
4150*
MANSUETO, S.
4814
MANZILLO, G.
4879
MARATKIA, Z.
4552
MARCADE, J.P.
4832
MARDAROWICZ, C
5044
MARENCO, G.
4840
MARES, A.J.
4599
MARGRAF, H.
4952*
MARGULIS, A.R.
3805
MARIN, A.W.
4578
MARINARI, U.M.
4118, 4119
MARKOV, J.G.
4035
MARKOW, J.G.
4030
MARKWARDT, J.
4249
MARLAND, P.
4775
MARQUAND, J.
4747
MARRIE, A.
4582
MARRIE, D.
4768
MARTA, J.B.
4287, 4288
MARTENS, R.A.
5108
MARTIN C.E.
4834
MARTIN, D.A.S.
4601
MARTIN, E.D.
5118
MARTIN, F.
4178
MARTIN, M.
4178
MARTIN, N.C.
5086
MARTIN, R.
4416
MARTINEZ, L.O.
4337

MARTINEZ-TORRES, C.
3806*
MARTINSON, J.
3864*
MARUYAMA, S.
5074
MARY, J.Y.
3839
MASCIARIELLO, S.
4836
MASELLI, J.P.
4497*
MASLINSKI, C.
4159
MASON, D.K.
5124
MASON,E.E.
4327
MASON, J.
3848
MASSA, C.
4644
MASSARANI, E.
4070
MASSARRAT, S.
4092
MASSELINK, B.A.
4517
MASSUN, P.
4243
MASTNAK, C.
4921
MASUTTI, A.
4956
MASZEWSKA-KUZNIARZ, K.
5045
MATHAN, M.
4172
MATHAN, V.I.
4172
MATHIEU, B
4610*
MATSUDA, Y.
4863, 4864
MATSUMOTO, T.
4385*
MATSUY, S.P.
3959
MATSUYAMA, S.
4662
MATTE, J.
4877, 4878
MATTERN, P.
4170
MATTY, A.J.
4637
MATUCHANSKY, C.
5114
MATUYOSHI, M.
4233
MATUYOSHI, T.
4233
MATVEEVAL, A.A.
4091
MATZKIES, F.
4157
MAUBEL, G.
4184*
MAURER, R.
4060
MAURER, W.
4397, 5059
MAURICE, P.
4877
MAY, VON D.A.
4548
MAYAT, V.S.
4984
MAYNARD, J.E.
5112
MAYOR, G.
4997
MAYR, W.W.
4000*
MAZANOV, G.S.
4560
MAZHENOV, A.S.
4211
MAZNEVA, L.M.
4338
MAZNILLO, G.
4846
MAZURIN, A.V.
4201
MAZZACCA, G.
3969
MAZZONI, G.
4828
MCALHANY, J.C., JR.
4390*
MCARTHUR, P.
4557
MCCAFFERY, T.D., JR.
4474
MCCAFFERY, T.D, JR.
5123
MCCALL, J.T.
3986
MCCAY, P.B.
4052
MCCLOY, R.E. JR.
3869*
MCCLURE, J.P.
4607*
MCDONAGH, A.F.
4005*
MCINNIS, W.D.
5081
MCLELLAN, J.S.
4866
MCMAHON, R.E.
4051
MCMANAMON, P.J.M.
4943*
MCMANUS, J.P.A.
4310
MCMULLEN, J.J.
4047
MCNAIR, A.
4498*
MCNEISH, A.S.
4602*, 4632
MECKELER, K.J.
4479
MEDEE, W.
4414
MEDINA, O.F.
5122
MEDKOVA, I.L.
3987
MEEKER, W.R. JR.
4244
MEEROFF, J.C.
3939*
MEEROFF, M.
4704
MEGUID, M.
4187*
MEGUID, M.M.
4576
MEHTA, J.M.
4499*
MEIER-RUGE, W.
4708
MEIJER, A. J.
4050
MEIJER, D.K.F.
3854
MEINERO, M.
4724
MEISSNER, R.
4908
MEICHIORRI, P.
3962
MELIS, M.
4828
MELKI, G.
4822
MELMAN, E.P.
4135
MELNIK, L.A.
4978
MELNYCHUK, D.B.
4022
MEMBRENO, A.
4187*
MENARDO, G.
4840
MENG, W.
4850
MENGE, H.
3815*, 3819*, 4628
MENKES, C.J.
5100
MENSORIO, C.
4549, 4585
MERCER, H.D.
4183
MERGUET, P.
3955
MESCIA, C.
4927
MESSE, A.A.
5122
MESSIAN, R.A.
4295
METREAU, J.M.
4798, 4942*
MEVES, M.
3888
MEYER-BURG, J.
4928, 4999, 5055

MEYER, C.
4574, 4582
MEYER,E.
4171
MEYER ZUM BUSCHENFELDE, K.H.
4880, 5184
MEYERBURG, J.
4261
MEYERS , M.A.
4647*
MEZEY, E.
3835
MICHEL, B.
5128
MICHELAZZI, L.
4118
MIDEJEAN, A.
3865*
MIEDERER, S.
4255
MIEDERER, S.E.
4218, 4257, 4531
MIGNECO, G.
4814, 4826
MIGNON, M.
5025
MIHATSCH, M.J.
4332
MIKELADZE, K.D.
5010
MIKHAILENKO, A.A.
4891
MILCH, H.
5030
MILLAT, R.
3963
MILLER, G.
4312, 4397, 5059
MILLER, H.H.
4717
MILLER, R.C.
5110
MILLER, S.S.
4308
MILLER, W.N.
3873*, 4299
MILNOV, O.B.
4909
MILONE, E.
4038, 4136
MILONOV, O.B.
4824
MILTON-THOMPSON, G.J.
3899
MING CHU, T.
4646*
MINH, V.T.
4577, 4586
MINKOWITZ, S.
4040
MINNE, H.
3953, 3954, 4494*
MINOGINA, N.A.
4801
MIRKOVITCH, V.
3819*
MIRONOV, O.N.
4989
MIRONOV, S.P.
4809
MIROSHNICKENKO, V.A.
4201
MISAKI, E.
4743*
MISHIN, V.
3999*
MISHRA, N.K.
3906
MISIEWICZ, J.J.
3899, 3908
MISKIEWICZ, H.
4323
MISRA, R.A.
5090
MISZ, M.
4461
MITCHELL, A.B.S.
5079
MITCHELL, C.G.
4901*
MITCHELL, J.H.
4949*
MITCHELL, R.L.
4319
MITOV, E.
4570
MITRA, M.N.
3853
MITTAL, V.K.
4705
MIURA, T.
4223, 4228, 4233, 4252
MIYA, T.S.
4058
MIYAI, K.
4000*
MIZUGAKI, J.
4114
MIZUNO, H.
4371*, 4476
MLYNCHIK, V.E.
4445
MOCANU, A.
4733
MODIGLIANI, R.
3839
MODRZEWSKA, R.
5044
MOERTEL, C.G.
4657*
MOGILEVSKAIA, I.A.
4820
MOLASKY, D.
4085
MOLINA-ESCOBAR, J.
4554
MOLINIE, C.
4894
MOLODENKOC, M.N.
4753
MONGES, H.
3878, 3895
MONNIER, J.P.
4567
MONTALI, R.J.
4195*
MONTI, G.
4021
MOORE, B.
5080
MOORE, E.P.
3868*
MOORE, J.G.
3932*
MOORJANI, S.
4915
MORAVEC, R.
4746
MORDUKOVITCH, K.T.
4147
MOREINO, M.S.
4753
MORGAN, D.B.
4709*
MORGAT, J.L.
3963
MORGENSTERN, L.
4272
MORIAU, M.
4938
MORIMOTO, T.
5074
MORIOKA, M.
4459
MORITA, A.
4496*
MORITZ, B.
4027
MORIUCHI, S.
4162
MOROKOVA, R.N.
4201
MOROZ, I.M.
4749
MORRIS, D.J.
3857
MORRIS, J.S.
4948*
MORRISON, A.B.
5121
MORRISON, K.E.
4085
MORRISSEY, J.F.
4473
MORSON, B.
5196
MORTON, J.J.
4663
MORUZZI, M.S.
4021
MOSAVY, S.H.
4732
MOSCHINSKI, D.
5007
MOSCOWITZ, A.
4174
MOSENTHAL, W.T.
4235
MOSEVITCH, T.N.
4145
MOSS, A.A.
4817
MOTOVIC, A.
4478

MOTYKA, G.
4139
MOUCHET, A.
4747
MOULE, Y.
4034
MOULT, P.J.A.
4857*, 4929
MOUNTAFIAN, J.P.
5019*
MOVCHUN, A.A.
4909
MOWAT, N.A.G.
4485*
MOZHAEV, H.A.
4097
MROCZKA, J.
3978*
MUCKE, D.
5103
MUELLER, R.F.
4680
MUGNIER, B.
4269
MUI, M.M.
4013*
MUKADA, T.
4345
MUKHOPADHYAY, A.K.
3867*, 4375*
MULL, R.H.
4148
MULLER, D.
4232, 4618
MULLER, W.A.
5135, 5140, 5141
MULLER-WEILAND, K.
4414
MUNCHOV, R.
4933
MUNOZ MONTEAVARO, C.
4927
MUNRO, A.
4444
MURAKAMI, K.
4229
MURANISHI, S.
3834
MURIEV, A.I.
4771
MURPHY, G.M.
4656*
MURPHY, G.P.
4646*
MURRAY, C.R.H.
5083
MUSSCHE, M.
4643
MUSSO, A.M.
4926
MYKHAILOVSKY, V.O.
4022
MYKONIATIS, M.G.
4124
MYNONOVA, V.M.
4112
NABSETH, D.C.
4497*
NACCARATO, R.
4903*
NADJ, G.
4234
NAFRADI, J.
4205
NAGARAJAN, V.
4861*
NAGASHIMA, K.
4662
NAIK, S.R.
4375*
NAIR, R.R.
4469
NAIR, V.
3790*
NAIRN, R.C.
4687
NAJARIAN, J.S.
4679
NAKAGIRI, S.
4863, 4864
NAKAJIMA, M.
4229
NAKAMURA, K.
4388*, 4496*
NAKANO, H.
4027, 4074, 4104
NAKANO, J.
3869*
NAKANO, S.K.
3869*
NAKASHIMA, H.
4025
NAKAYAMA, K.
4352
NAKAYAMA, M.
4352, 5105
NALBONE, G.
3975*
NALIVKINA, A.I.
3838
NANNI, G.
4118, 4119, 4578
NARAYAN, K.A.
4047
NARULA, I.M.S.
4686
NASATKIN, I.U.M.
4809
NASKE, P.
3941*
NATARAJAN, T.K.
4940*
NAUBER, G.
4249
NAUDY, B.
3878
NAUER, E.
4417
NAVARRO, J.
4606*, 4920*
NAWRATH, G.D.
3951
NAZARIAN T.
5078
NEBEL, O.T.
4295
NECHAEVA, N.V.
3914
NEEDHAM, C.D.
4485*
NEGRO, P.
4946*
NEGROTTO, J.
5061
NEILSEN, O.V.
4498*
NEKHAY, K.KH.
4748
NELSON, J.A.
4300, 4311
NELSON, O.
4556
NELSON, R.
4381*, 4656*
NEMOURS-AUGUSTE, S.
4309, 4313
NEMSMANN, B.
4678
NESTERENKO, H.A.
4096
NESTERINA, L.F.
5073
NESVETOV, A.M.
4446
NEWCOMBE, R. G.
4105
NEWCOMER, A.D.
4641
NEWMAN, A.
3899
NEWTON, C.R.
4721
NEZAMIS, J.E.
4501*
NG, K.H.
4555
NGUYEN, T.T.
4055
NIANG, I.
5147
NICAK, A.
4538
NICHOLLS, D.M.
4068
NICOLI, N.
4160
NIELSEN, V.
4791*
NIHEI, T.
3796
NIHOUL-FEKETE, C.
4547
NIKELOV, N.A.
3884
NILSSON, F.
3943*
NILSSON, G.
3966
NIMMO, W.S.
4377*
NIND, A.P.P.
4687
NIPPER, T.
4186*

NISEVICH, N.I.
4890
NISHIGORI, S.
4233
NISHIOKA, K.
4682
NISKI DE RYDEL, R.
5061
NITZ, I.
5103
NIXON, H.H.
4720
NORMANN, E.
4054
NOGUEIRA, C.E.D.
4742*
NOGUERIA, C.E.D.
4737*
NOLAN, N.G.
4200*
NOMURA, T.
3916
NORGRADI, E.
4351
NORMAN, A.W.
3847, 3853
NORMAN, M.R.
4088
NORRIS, C
3855
NORTHFIELD, T.C.
4941*
NORTON, L.W.
5085
NOSOV, I.S.
4985
NOVIS, B.
3942*
NOVOKSHONOV, A.A.
4890
NOVOZHILOVA, Z.I.
4614
NOWAK, W.
3985
NOZDRACHOVA, L.I.
4076
NOZDRINA, R.A.
4215
NUESCH, H.J.
4659
NUMA, S.
4044
NUNES, A.
4737*
NUTI, M.
4907
O,BRIENS, R.J.
4151*
O LEARY, J.P.
4583
OBERFIELD,, R.A.
4781*
O'BRIEN, P.
3938*
O'BRIEN, R.
4598
OBTULOWICZ, W.
3931*
OBUKHOV, V.A.
5012
O'CONNOR, J.
5178
OCZKO, B.
4249
ODIEVRE, M.
4785*
OGATA, H.
3916
OGATA, K.
4114
OGATA, M.
4114
O'HARA, E.T.
4497*
OHISHI, H.
4223, 4228, 4233, 4252
OHRADKA, B.
4746
OHRADKA, B
4746
OHTANI, Y.
4233
OJDANIC, Z.
4234
OKABE, S.
4388*, 4391*, 4496*
OKAMURA, W.H.
3853
OKIGAKI, T.
3786*
OKOLSKA, G.
3824
OKUBO, T.
4682
OKUDA, Y.
4863, 4864
OLAH, B.
5030
OLBERMANN, M.
4232, 4618
OLCEN, P.
5127
OLDAK, B.
4596
O'LEARY, J.P.
4001*
OLIPHANT, M.
4647*
OLIVIER, L.J.
5153
OLSINA, J.
5042
OLTMANNS, D.
5066
ONDREICKA, R.
4046
ONOFREI, T.
4733
OPPITZ, K.H.
5091
ORESHKOV, T.M.
4427
ORIANI, G.A.
4812
ORIMA, H.
4391*
ORTEGA VEGA, J.
4830
ORTIZ URDIAN, G.
4892
OSHIMA, H.
4466
OSHITA, Y.
4026
OSIS, D.
3855
OTAWA, K.
4706
OTTE, J.B.
4938
OTTE, M.
4247
OTTENI, F.
4574, 4582
OTTENJANN, R.
4203, 5043
OTTO, H.F.
4611*
OTTO, H.H.
4939
OTVOS, I.
4910
OUIMET-OLIVA, D.
5109
OVCHININSKY, N.N.
4984
OVCHINNIKOVA, L.P.
4142
OVECKA, M.
4046
OVERHOLT, B.F.
4237
OWEN, O.E.
3937*
PACESCO, E.
4961
PACZOS-CHADYMA, E.
5050
PAGANELLI, F.
4558
PAGES, R.
4653*
PAIGE, D.
4644
PALA, V.
4119
PALAGIANO, V.
4562
PALAMARCHUK, V.P.
4831
PALASZEWSKA, M.
4153*
PALIARD, P.
4727
PALMA, F.
4409
PALTANAVICHIUS, K.I.
4317
PANCHENKO, L.F.
4076
PANSU, D.
4155*
PANTSYREV, Y.M.
4400

PAOLAGGI, J.A.
4269
PAPACHARALAMPOUS, N.X.
4124
PAPAMIO RUIBAL, A.
5065
PAPP, L.
4461
PAPP, M.
4166*
PAR, A.
4910
PARA, M.F.
4476
PARBHOO, S.
4804
PARBRIDGE, W.M.
3811*
PARE, W.P.
3921*
PARIS, J.
4965, 4993
PARIS, J.C.
4914*
PARK, C.
4694
PARK, D.H.
3930*
PARKS, M.P.
4286
PAROLI, E.
4120
PARSONS, C.
3935*
PARSONS, M.F.
3972
PARTENSKY, C.
5190
PASTORE, G.
4897
PATARAKINA, A.A.
4231
PATEY, A.L.
4629
PATINO, J.
4792*
PATORSKA-MACH, E.
5044
PATRASSI, N.
4946*
PATSIORA, M.D.
4031
PATT, D.J.
4648*
PATTISON, C.P.
5112
PAULSEN, G.
3939*
PAUMGARTNER, G.
3807*, 3994*, 5003, 5009
PAUTRIZEL, A.N.
4170
PAUTRIZEL, R.
4170
PAVLOV, K.A.
4224
PEACU, Y.
4920*
PEARSE, A.G.E.
5117
PEDERSEN, T.
4412
PEES, H.
4848
PEIPER, H.
4304
PEIPER, H.J.
4306, 4759
PELIZZO, M.R.
4956
PELKONEN, P.
4603*
PELLEGRINI, S.
4828
PELLERIN, D.
4547
PELLONI, S.
4423
PELOQUIN, A.B.
4349
PENAUD, J.
5019*
PENCHARZ, P.B.
5096
PENDSE, A.K.
4686
PENKA, W.E.
4701
PENKOV, L.K.
4445
PERCY, J.S.
5170*
PEREZ SOTELO, R.
4563
PERGAM, C.J.
4431
PERGOLIZZI, R.
5191
PERISI, P.
3902, 4587
PERISIC, R.
5009
PERISSAT, J.
4959
PERITTI, P.
4123
PERRET, G.
3926*
PERRINO, G
3971
PERROTTO J.L.
4871*
PERSIGEHL, M.
4735
PERSSON, B.
5016*
PERZIN, K.H.
4364, 4541*
PESCATORI, M.
4578
PESCH, H.J.
4157
PESCHL, L.
4103
PETER, B.
4518
PETER, P.
4515
PETERS, R.L.
4780*
PETERSEN, N.J.
5112
PETROCCHI, G.
4549
PETRONIO, R.
4160
PETROV, V.I.
4771
PETROVIC, Z.
5134
PETROVSKAIA, V.G.
5075
PETRYKA, Z.J.
4174
PETTIROSSI, O.
4038, 4136
PFEIFFER, E.F.
4226
PFENNINGER, E.
4458
PFENNINGER, K.
4841
PHILIPPEN, R.
5036
PHILLIPS, J.C.
3818*
PIANOV, R.P.
5071
PIANTE, M.
4586
PICCININO, F.
4846, 4879
PICHLMAYR, R.
4522, 4661
PICKFORD, I.R.
4508*, 4525
PIEKARSKI, G.
5133
PIETKIEWICZ, K.
5046
PIGLIUCCI
4895
PIHL, E.A.V.
4687
PILLON, M.
4727
PIMPARKAR, B.
4713
PIMPARKAR, B.D.
4499*
PINCHON, J.
3984
PINCHON-MARSY, S.
4592
PINOCHON-MARSY, S.
3984
PIOCH, E.
4969
PIRK, F.
4256, 4552
PIROLA, R.C.
4154*
PIROZYNSKI, W.
4339

PIZINGER, R.J.
4287, 4288
PLAKHOTINIUKOVA, V.V.
3991
PLAMINEIAK , L.
4899*
PLANETA-MALECKA, I.
5047
PLESSIER, J.
4520
PLEVY, D.
4329
PLEWA, T.
4462
PLIAM, M.B.
4722*
PLOMTEUX, G.
4872*
POCURULL, D.W.
4183
POGONOWSKA, M.J.
4333
POGORELOV, I.F.
4036
POINTER, H.
4190*
POINTER, R.H.
4080
POIRAT, F.B.
4653*
POKORA, J.
4802
POKROVSKII, A.A.
3797, 4041
POKROVSKII, G.A.
4238
POLIAKOV, E.L.
3893
POLISCHUK, A.S.
4016
POPIELA, T.
4505*
POPOVA, I.V.
4073
POPOVICI, G.G.
4733
POPVICI, G.I.
4733
PORETZ, D.M.
4694
POROKHNIAK, L.A.
4137
PORTA, E.A.
3804
POSKITT, E.M.E.
5023
POSNER, B.I.
4099
POST, C.
5078
POSTIGLIONE, G.
4725
POSTLOV, M.P.
4750
POSTUPAEV, V.V.
4077
POTET, F.
3798, 3799, 5118
POUYET, M.
4024
PRADERI, L.A.
5062
PRASAD, A.
4540*
PRATHAP, K.
4555
PREISIG, R.
4191*
PRELLWITZ, W.
4232, 4618
PRESCOTT, L.F.
4377*
PRESTELE, H.
4157
PREZIOSI, M.
4573
PRIMACK A.
4794*
PRIOR, A.
5176
PRISTAUTZ, H.
5001
PROKOPOVA, I.V.
4201
PROSS, E.
4752
PRUITT, B.A., JR.
4390*
PRYADKIN, V.V.
4983
PRZYWARA, S.
4462
PUCHKOV, J.
4224
PUKHAEVA, R.G.
4141
PUMPHREY, H.
4795*
PUNTILLO, E.
4991
PUSTOVALOVA, L.M.
4111
PYSZ, H.
4462
QUAN, S.G.
3786*
QUATTROPANI, S.L.
3787*
QUERTILLIER, D.
4692
QUESSADA, M.H.
3896
RABEN, N.A.
4762
RABENDA, C.
4905*
RABES, H.M.
4020
RACHAIL, M.
4189*
RADULESCO, D.
4961
RAIA, A.
4651*
RAJU, G.C.
4389*
RAMACH, K.R.
4593
RAMBAUD, J.C.
5114
RAMDEO, I.N.
4686
RAMOS, R.
5081
RAMPAL, P.
4407
RANEK, L.
4791*
RANGACHARI, P.K.
3923*
RAPP, W.
4443
RAPTIS, S.
4226
RASENACK, U.
4115
RASMUSSEN, K.
4498*
RASSER, Y.M.
4064
RASTELLI, M.
5037
RASTOGI, M.
5090
RATH, R.
4619
RATHELOT, J.
3975*
RAU, R.
4339, 4841
RAUCHENBERGER, B.
4439
RAULT, D.
4914*
RAUTENBACH, M.
4608*
RAVITCH, M.M.
4595
RE MINE, W.H.
4722*, 4728
READ, A.E.
4609*
RECKER, R.R.
3845
REDDY, D.B.
4389*
REDEY, B
5030
REDING, R.
3985
REED, J.D.
3922*, 3934*, 3944*, 3973
REED, W.D.
4901*
REEDER, D.D.
3924*, 5093
REFAI, F.M.
5087
REGENT, M.
4416
REHBEIN, F.
4330
REICH, K.
4321

REICHART, E.
4366*
REICHEL, K.
4527
REICHEN, J.
3807*
REID, D.R.K.
4945*
REIFMANN, V.K.
4280
REILLY, R.W.
4289, 4658
REINER, J.M.
4129
REISENAUER,
4623
REMACLE, J.P.
3895
RENDA, A.
4836
RENNER, F.
4698
RENNERT, D.M.
4934
RENZY, M.
3806*
RERAT, A.
3814*
REVIL, M.
4416
REVITCH, G.G.
4147
REVUTSKY, B.I.
5053
REY, F.
3826
REY, J.
3826
REY, R.
4792*
REYES, C.N.
5150
REYNOLDS, D.G.
4037
REYNOLDS, T.B.
4780*
RICARDES, M.I.
4193*
RICCOUR, C.
4547
RICE, R.P.
5169*
RICHARDS, M.P.
4069
RICHARDSON, A.L.
4000*
RICHARDSON, C.T.
4488*, 4491*
RICHARDSON, J.D.
5081
RICHTR, C.
4242
RICK, W.
3961, 4398, 4399
RIECKEN, E.D.
3815*, 4628
RIEDE, U.N.
4053, 4054, 4064
RIEGLER, G.
4812
RIGBY, C.
4557
RINSTEN, A.
4378*
RINALDO, J.A.
4361
RIOTTOT, M.
4167*
RIPAULT, J.
4653*
RIPPE, D.F.
4066
RIQUELME, P.
4563
RITCHIE, J.K.
4719
RITCHIE, W.P., JR.
3844
RITCHIE, W.P. JR.
4386*
ROBAUSCH, T.
4053
ROBBE, K.
5103
ROBERT, A.
4501*
ROBERTSON, A.F.
5113
ROBINSON, D.A.
3894
ROBINSON, J.W.L.
3819*
ROCCHETTI, P.
4923*
ROCHNA VIOLA, E.M.
4926
RODGERS, J.B.
4151*
RODINOV, V.M.
4018
RODRIGUEZ, A.
5082
ROGER, J.
4965
ROGERS, I.M.
4945*
ROGOZ, J.
4848
ROGOZINA, E.M.
3948
ROHR, H.P.
4064
ROHRMANN, C.A.
4294
ROKHLIN, E.A.
4208, 4446
ROLLAND, J.M.
4687
ROLLES, C.J.
4602*
ROLLINS, L.D.
4183
ROLLO, A.J.
4687
ROMAN, D.
4874*
ROMANI, F.
4724
ROMMELSHEIM, K
4074
RONAIL, A.
3890
ROSA, G.
4818
ROSANOV, A.
4075
ROSANOV, Y.M.
4079
ROSARIUS, C.
4202
ROSCH, J.
4184
ROSCH, W.
4434, 4451, 4455, 5185
ROSELLEN, E.
4763
ROSENBUSCH, G.
4275
ROSENMAN, L.D.
4691
ROSENTHALE, M.E.
4182
ROSENWEIG, N.S.
4161
ROSIEK, B.
4884, 4885, 4886, 4887
ROSOVA, E.I.
3962
ROSSIER, M.
3809*
ROSSIPAL, E.
4627
ROSSNER, J.A.
4839
ROSSOUW, J.E.
4858*
ROTHFELD, B.
4644
ROTHMAN, S.S.
3809*
ROTHMUND, M.
4514
ROUCAYROL, J.C.
5100
ROUSSEAU, C.
4877, 4878
ROUSSEAU, V.
5132*
ROUSSILLE, M.
4727
ROVIERA, C.
4558
ROYER, G.
4381*
ROYER, P.
4547
ROZANOV, B.S.
4208
ROZE, C.
3979*, 3980*
ROZENTAL, R.L.
4030
RUBASHNAYA, I.
4984

RUBIN, R.J.
4689
RUCINSKI, J.
4506*
RUCK, B.J.
5014*
RUDIER, M.
4393
RUDKOWSKI, Z.
4905*
RUGE, W.
5041
RUGGIERO, G.
4879
RUIZ DE AGUIAR GONZALEZ, A.
4700
RUIZ MAURY , A.
4811
RUIZ, R.
4475
RUOFF, H.J.
3970
RUPPIN, H.
3887
RUSAKOV, V.I.
4111
RUSSELL, A.S.
5170*
RUSSELL, R.I.
4376*
RUTORYAN, E.S.
5069
RUTTAS, M.
4481
RUWART, M.J.
3977*
RYAN, D.
4083
RYBAKOVA, G.S.
4597
RYSS, E.S.
3948
SABA, V.
3902
SABO, D.
4932
SACCO, M.
4565
SACHAR, D.R.
5163*
SACHS, G.
3842, 3843
SACQUET, E.
4167*
SADAN, N.
4478
SAEGESSER, F.
4693, 4695
SAEGUSA, J.
5105
SAFAIE-SHIRAZI, S.
4327, 4788*
SAFIESHIRAZI, S.
4301
SAFRANEK, V.
4970
SAGNELLI, E.
4846, 4879
SAHEL, J.
4765
SAILER, R.
5007
SAINTCYR, M.
4577
SAITO, T.
3796, 4230
SAKIBOV, I.A.D.
5029
SAKODA, K.
4957
SALDUCCI, J.
3878, 3895
SALEMBIER, Y.
4965, 4993
SALMASO, G.C.
5005
SALMON, J.A.
3967
SALTER, R.H.
4516
SALVA, J.A.
4600
SALVATI, E.P.
4689
SALWAKUBASIK, W.
4851
SAMOKHINA, Z.F.
5076
SAMPLINER, J.E.
5015*
SAMSIOE, G.
4955*
SAMSO, G.
4993
SAMSON, E.I.
4512*
SAMSONOV, V.A.
4432
SAMUELI, F.
4120
SANCHEZ SALGADO, G.
4892
SANCHEZ, VENEGAS, S.
4264
SANDBLOM, P.
4695
SANDERS, D.J.
3934*, 3973
SANDERSON, C.R.
4482*
SANDOR, A.
4182
SANDRITTER, W.
4053
SANKALE, M.
5147
SANO, R.
4387*
SANTAGOSTINO, C.
4227
SANTINI, R.
3901
SANTULLI, T.V.
5017*
SAPELKINA, I.M.
4400
SARACINO, F.
4585
SARDA, L.
3975*
SARIN, G.S.
5090
SARLES, H.
4730, 4765
SARLES, J.C.
3865*
SARRIS, M
4669
SARTOR, V.E.
5170*
SASAJIMA, K.
4387*
SASIAK, K.
4159
SASSA, R.
4837
SATGE, P.
4855
SATO, A.
3885
SATO, E.
4345
SATO, T.
3870*, 4411
SATO, Y.
3885
SAUBIER, E.C.
5190
SAUNDERS, S.J.
4858*
SAVA, G.
3898
SAVAROVSKII, A.I.
4338
SAVILAHTI, E.
4603*
SAVILLE, P.D.
3845
SAVINA, M.I.
4147
SAVISAAR, E.
4481
SCALLEN, T.J.
4043
SCHACT, U.
3961, 4398, 4399
SCHAEFER, K.
4467
SCHALLY, A. V.
3944*
SCHANG, J.C.
3879, 3898
SCHARFETTER, H.
4341
SCHEININ, T.M.
4353
SCHENK, E.A.
4604*
SCHENKEN, J.R.
4795*
SCHENKER, S.
4793*
SCHERSTEN, T.
4797*

SCHEUER, P.J.
4948*
SCHEURER, U.
5187
SCHEURLEN, P.G.
4848
SCHICKEDANZ, H.
4546, 4584
SCHIER, J.
4532
SCHILKOW, P.
4958
SCHILLER, W.R.
4165*
SCHIMMEL, E.M.
4497*
SCHINDLER, D.
5041
SCHIRALDI, D.
4897
SCHIRMER, G.
5175
SCHLAAK, M.
4665
SCHLAUT, J.
5170*
SCHLEGEL, E.
4149*
SCHMID, M.
4395
SCHMID, R.
4005*
SCHMIDT, H.
4759
SCHMIDT, K.
4620
SCHMIDT-KESSEN, W.
5097
SCHMIDT, P.
4190*
SCHMIDT-WILCKE, H.A.
3951, 4429
SCHMITT, M.G. JR.
4666
SCHMITZ, J.
3926
SCHNELL, R.C.
4058
SCHNELLBACHER, E.
4763
SCHOFIELD, P.F.
4951*, 5193
SCHOLL, A.
4620
SCHOLZEL, F.
4779
SCHOMERUS, M.
4589
SCHONE, S.
4608*
SCHOOLMEESTERS, J.F.P.V.
4259
SCHRAMM, E.
4438
SCHUEFEL, W.
5056
SCHULTHEISS, H.R.
5009
SCHULTZ, H.J.
4616
SCHULTZ, S.G.
3856
SCHULTZE, B.
4060
SCHULZ, H.J.
4773
SCHULZ, K.
4803
SCHULZE, D.
3941*
SCHUMAN, L.
4581
SCHUMPELICK, V.
4439, 4454, 4464
SCHUSTER, P.
3941*
SCHUSTER, R.
4419
SCHUTT, A.J.
4657*
SCHWARTZ, I.S.
4186*
SCHWARZ, H.P.
3994*
SCHWARZ, K.
4071
SCHWEMMLE, K.
4254, 4451
SCHWESINGER, G.
4964
SCHWESINGER, W.H.
4210
SCHWILDEN, E.D.
4668
SCHWORER, I.
4261
SCOTT, R.M.
4185
SCRIMSHAW, N.S.
5096
SCWACHMAN, H.
4544*
SEAH, C.S.
4268
SEAH, P.P.
4639
SEASHORE, J.H.
4853
SEETHARAM, B.
4043
SEIFERT, E.
4274, 4743*
SEIFERT, G.
4104
SEILER, P.
4997
SEITS, I.F.
4173
SEKINE, T.
3940*
SEMONOVA, N.F.
4156
SENIUTOVICH, V.F.
4770
SENOO, A
3796
SENOR URIA, C.
4563
SEPULVEDA, F.V.
3819*
SEREBRIAKOVA, N.N.
4231
SERGEEV, S.I.
4988
SERGIENKO, V.S.
4980
SERNKA, T.J.
3950
SERRA, E.
4123
SESSOMS, C.
5159*
SETNIKAR, I.
4070
SETTE, J. JR.
4935
SEWING, K.F.
3970
SEZAKI, H.
3834, 3860
SEZILLE, G.
4914*
SHABANOV, A.N.
5010
SHAH, B.
4343
SHAH, H.C.
4011*
SHAH, P.N.
4507*
SHAKHBAZIAN, A.A.
4715, 4716
SHALIMOV, A.A.
4303
SHALIMOV, S.A.
4267, 4303
SHALYGINA, N.
5102
SHALYGINA, P.B.
5098
SHAMANOV, A.K.H.
4251
SHANBOUR, L.L.
3925*
SHAPKIN, V.S.
4748, 4827
SHARSHAVITSKY, G.A.
4432
SHATNEY, C.H.
4740*
SHAW, B.
3944*
SHCHERBAKOV, A.A.
5052
SHEEDY, P.F. II
5022
SHEERIN, H.E.
4152*
SHEHATA, R.
3795
SHEINER, H.J.
3886, 4526
SHENYAKIN, G.L.
4816

SHENGELAIA, T.F.
4445
SHEPHERD, D.M.
4502*
SHERBANIUK, R.W.
5170*
SHERENI, P.
4932
SHERLOCK, P.
4384*, 4688
SHERLOCK, S.
4857*, 4948*
SHERMAN, J.O.
5018*
SHERWOOD, C.E.
4545*
SHEVCHENKO, B.I.
4536
SHEVCHENKO, V.S.
4134
SHEVCHUK, M.G.
4770
SHIBATSUJI, H.
4223, 4228, 4233
SHIBATUZI, H.
4252
SHIMADA, F.
3885
SHIMAMOTO, K.
4383*
SHINER, M.
4633
SHINKARENKO, N.I.
4201
SHIOMI, Y.
4863, 4864
SHIRATORI, T.
3940*
SHIU, M.H.
4819
SHLAPAK, I.P.
4097
SHLIGIN, G.K.
3989
SHLYGIN, G.K.
3990
SHMONINA, V.M.
4023
SHOEMAKER, R.L.
3843
SHOLTS, N.F.
4031
SHONS, A.R.
4679
SHORE, J.M.
4272
SHORTSINGER, A.G.
4031
SHRIVER, D.A.
4182
SHTUTMAN, T.M.
4078
SHUBIN, B.M.
4977, 5069
SHUMANOVA, T.I.
4979
SHUNGAWA, M.
4223
SHUSTEROV, M.I.
4446
SHVESITSER, E.A.
4982
SIAI, D.J.
4092
SIANESI, M.
5006
SIDORENKO, V.I.
4400
SIEBERMANN, R.
4458
SIEDEK, M.
4257
SIEGERS, C.P.
3836
SIEPPEL-MACIEJEWSKA, K.
3978*
SIES, H.
4002*
SIESS, E.A.
4095
SIEWART, R.
4318, 4403
SIEWERT, R.
4304, 4306
SIGDESTAD, C.P.
4185
SILEN, W.
3938*
SILINA, E.D.
5072
SILK, D.B.A.
4605*, 5083
SILL, U.
3985
SILVERBERG, M.
5191
SILVERMAN, J.A.
3857
SILVESTRI, F.
4038
SILVIS, S.E.
4294
SIMMONDS, R.C.
3804
SIMON, R.
4794*
SIMPSON, J.G.
4444
SINGER, P.
4844
SINGH, G.
3875*
SINIAEV, V.P.
4771
SINNIAH, D.
4198*
SIPES, I.G.
4057
SIRINEK, K.R.
4918*
SISOIEV, I.U.A.
3990
SIVANAGIMANI, R.
4389*
SIVKOV, I.I.
4852
SKALA, I.
4256, 4754
SKALA, J.
4754
SKALA, L.
4256
SKARLOSH, V.M.
4132
SKILLMAN, J.J.
3938*
SLAFF, G.F.
3946*
SLAVIN, G.
4913*
SLEE, R.G.
5106
SLEGERS, J.F.G.
3949
SMALE, L.E.
5137
SMALLWOOD, R.
4530
SMIRNOV, K.V.
3987
SMIRNOVA, S.V.
4215
SMIRNOVA, V.N.
4018
SMITH, E.H.
4199*
SMITH E.I.
5173*
SMITH, F.H.
3851
SMITH, I.S.
4511*, 5178
SMITH N.
4328
SMULEWICZ, J.J.
4359
SMY, J.R.
3922*
SNEDDEN, W.
4654*
SNOP, S.
3915
SOBBE, A.
4218, 5175
SOERGEL, K.H.
3877
SOINE, L.
5104
SOKAN, I.
3896
SOKOLEWICZ, E.
4870*
SOKOLOBA A.S.
4231
SOLDANI, G.
3863*
SOLLBERGER, H.
4934
SOLNTSEVA, A.A.
4215
SOLOMIANSKII, A.E.
4537
SOLTAS, J.G.
4066

SOMANI, P.N.
5154
SOMMER, H.
4246
SOMMER, H.M.
3825
SOMOVA, E.P.
4403*
SONDOR, A.A.
4035
SONENBERG, M.
4384*
SORRANTCI, CH.
3962
SORENSON, M.B.
4498*
SORGATO, K.
4121
SORIANO, A.
4342
SORINSON, S.H.
5040
SORINSON, S.N.
4991
SOTNICHENKO, B.A.
4748
SOTOS, J.
5113
SOUCHARD, M.
3979*, 3980*
SOULLARD, J.
4242
SOUMARMON, A.
3947
SOUSSALINE, M.
5183
SOYKA, L.F.
3852
SPARKMAN, J.
4333
SPAY, G.
4542*
SPEDDING, M.
3907
SPEIRS, R.L.
3911
SPEISER, P.
3821
SPENCER, H.
3855
SPENCER, J.
4441
SPENNEY, J.G.
3843
SPIEGEL, P.K.
4235
SPRECHLER, M.
4967
SRIKANTAIAH, M.V.
4043
SRIVASTAVA, P.K.
5154
SRIVASTAVA, P.N.
5090
ST. STENDER, H.
4274, 4743*
ST. YVES, C.
5132*
STACHER, G.
3941*
STADELMANN,
4255
STADELMANN, O.
4218, 4257, 4531
STADLER, G.A.
4433
STAHL, A.
4518
STAHL, E.
4950*
STAJIC, M.
5139
STAKEBERG, H.
4797*
STALLKAMP, B.
4101
STALPORT, J.
4692, 5068
STAMOULAS, O.
4346
STANDAERT, L.
4370*
STARKER, H.
3941*
STARKLOFF, G.B.
4593
STASTNA, R.
4754
STAUBER, R.
5000
STEF, J.J.
4299
STEFANENKO, J.F.
4224
STEFANINI, P.
4946*
STEFANOV, A.V.
3992
STEFANOVIC, Z.
4683
STEFANOW, W.
4958
STEFANSKA-SULIK, E.
4596
STEFFEE, W.P.
5096
STEGMANN, S.
4813
STEIN, H.D.
4789*
STEIN, T.
4952*
STEINBERG, D.M.
4517, 5164*, 5182
STELLA, G.D.
4956
STELZNER, A.
4171
STELZNER, F.
4314, 4673
STEMPIEN R.
4906*
STENGER, V.G.
3787*
STEPHENS, D.H.
4200*
STERKY, G.
5016*
STERN, P.
4108
STERNBERG, S.S.
4553
STEVENS, D.P.
4687
STEWARD, J.S.
4631
STEWART, E.T.
4817
STOCKER, H.
4426
STOCKINGER, F.
4568
STODDARD, C.J.
4530
STOKES, P.L.
4636
STOPIK, D.
4098
STORCH, W.
4921*
STORCK, D.
5070
STORCZ, J.
4910
STOROZHAKOVA, IA.
4860*
STOROZHUK, P.G.
3991
STRADNIEKS. S.
4133
STRAIN, W.H.
5128
STRANADKO, E.F.
4445
STRASBERG, S.M.
4009*
STREKALOVSKII, V.P.
4238
STREMPLE, J.F.
4379*
STROBER, W.
5159*
STROHMEYER, G.
4806
STRUBELT, O.
3836
STRUCHKOV, I.U.V.
4211
STUBY, K.
4997
STUHLER, T.
4403
STURNIOLO, G.C.
4903*
STUTTE, H.
4665
SUC, J.M.
5067
SUCHKOV, A.V.
4911
SUCHNICKA, R.
4714
SUDAKA, P.
4407

SUTGIMURA, T.
4387*
SUKIRNO, M.S.
4911
SULLIVAN, M.
4340
SULOCKA, J.
5048
SULSER, H.
4684
SUMNER, H.W.
4655*
SUNAGAWA, M.
4228, 4233, 4252
SUNG, J.L.
4815
SURI, K.
3804
SUSSMAN, M.
4835
SUTER, H.R.
4397
SUZUKI, N.
4662
SUZUKI, O.
4459
SUZUKI, T.
3881, 4394
SUZUKI, Y.
4391*
SVENSSON, S.O.
3956, 3965
SVIDRO, A.
5030
SVIRIDOV, N.K.
4106
SVYSTUN, T.I.
3959
SWADZBA, S.
4968
SWAMINATHAN, S.P.
4172
SWAN, K.G.
4037
SWEETMAN, A.J.
3907
SWENSON, S.A.
4795*
SYDORENKO, O.S.
4087, 4089
SYLONOVA, N.V.
4022
SZABO, A.
4461
SZAFRAN, Z.
4505*
SZARVAS, F.
4849
SZCZGIEL, B.
4506*
SZELAG, O.
4870*
SZENTGALI, J.
5030
SZPALOWSKI, J.
4456
SZYKZKO, R
5044

TABRIZIAN, N.
4693
TACZANOWSKI, W.
4177
TADAION, A.
4732
TADOLINI, B.
4021
TAENZER, V.
4278
TAGAWA, S.
4863, 4864
TAKAGI, K.
3870*, 4388*, 4391*, 4496*, 4500*
TAKAGI, M.
4114
TAKAHARA, S.
4114
TAKAHASHI, A.
4252
TAKAHASHI, H.
4223, 4228, 4233, 4252
TAKAMURA, U.
4459
TAKANO, S.
3881
TAKASHASHI, E.
4442
TAKASHASHI, T.
4922*
TAKAYANAGI, I.
3870*
TAKAYANAGI, K.
4358
TAKETA, K.
4014*, 4061
TAKEUCHI, J.
5101
TAKEUCHI, K.
4388*, 4496*
TALAN, M.I.
3893
TALIANO, V.
4893
TALLERO, G.
4039
TALMAGE, R.V.
3804
TAMEDA, Y.
4863, 4864
TAMM, A.
4481
TAMURA, T.
5105
TAN, C.L.
4758
TANAKA, H.
4391*
TANAKA, M.
4682
TANAKA, N.
4387*
TANAKA, Y.
5092
TANGHE, W.
4551

TASHIRO, S.
4706
TASHKINOV, V.T.
4988
TASLER, J.
3931*
TASSINI, T.
4279
TATEO, R.
4724
TAUBERT, W.
4250
TAUSCH, V.
4933
TAUXE, R.V.
4509*
TAUXE, W.N.
4782*
TAYLOR, C.W.
4019
TAYLOR, J.G.
3894
TEDESCO, F.J.
4655*, 5013*
TEICHMANN, W.
4787*
TEIXIDOR, H.
4647*
TELNOV, Y.A.
3838
TEODORESCU, M.
5122
TERADA, M.
4863, 4864
TERENTIEVA, V.B.
4107
TERENTIEVA, V.G.
4090
TEVORADZE, L.SH.
4028
TEZUKA, F.
4922*
THATCHER, J.
4105
THEODOROPOULOS, G.
4664*
THERY, D.
4966
THIENPONT, L.
4643
THILLIER, J.L.
4521
THJODLEIFSSON, B.
4483*, 4502*
THOELKE, H.
4844
THOMAS, B.J.
4866
THOMAS, G.
4574
THOMAS, M.
4574
THOMAS, W.A.
4129
THOMPSON, H.
4638, 5181
THOMPSON, J.C.
3924*, 5093

THOMPSON, N.W.
4790*
THOMPSON, W.M.
5169*
THOUVENOT, J.
5019*
THOUVENOT, J.P.
4741*
THURNHERR, N.
4332
THUY, L.P.
4055
TIET, W.L.
4543*
TILAK, T.B.G.
4961*
TILBURY, T.
3804
TIRIBELLI, C.
4117
TITCHEN, D.A.
3919
TITOVA, L.S.
4231
TITOVA, V.G.
4110
TODOROV, T.N.
4063
TOGEL, H.
4996
TOGUZOV, R.T.
4029
TOKUNAGA, A.
4387*
TOLBERT, M.E.M.
4080
TOLEDO-PEREYRA, L.H.
4740*
TOMASI, V.
3995*
TOMASZEWSKI, J.
4802
TOMFORD, N.R.
4919*
TOMITA, T.
3871*
TOMOKUNI, K.
4114
TOMPA, G.
4461
TON THAT, H.
5067
TONGIO, J.
5070
TONISSEN, J.
5026
TONKOVIC, S.
5019*
TOOTH, E.
4351
TOPUZOV, E.G.
4447
TORIGATA Y.
3885
TORRES VERA, M.T.
4811
TOTCHASOVA, G.V.
4215
TOTH, I.
4849
TOULOUKIAN, R.J.
4853
TOUSSAINT, M.
4692
TOWE, J.
4260
TOWNELY, R.R.W
5014*
TOYE, D.K.M.
4630
TRAPNELL, J.E.
4777, 4778
TRAVKIN, I.U.A.
5012
TREADWELL, C.R.
3974*
TREE, M.
4663
TREMBLAY, M.
5132*
TRESHCHYNSKYI, A.I.
4097
TRETIAKOV, A.M.
5011
TRIER, R.
4397
TRIPI, S.
4814, 4826
TROESCH, V.
4540*
TROITSKAIA, V.B.
3882
TROMPETER, G.
4505*
TRUELOVE, S.C.
5160*, 5161*
TRUSOV, V.V.
4427, 4816
TSCHEN, H.
4196*, 4684
TSCHEN, H.H.
4415
TSIRONIS, A.G.
4124
TSITSISHVILI, TS.D.
4028
TSUJITA, E.
4863, 4864
TSYNKALOVSKII, I.B.
3991
TSYRLOV, I.
3999*
TUCKER, A.S.
5104
TUCKER, P.C.
5131*
TUCZEK, H.V.
4020
TUNG, L.C.
4101
TURCHEN, B.
4085
TURGANBAEV, A.
4348
TURNBULL, M.J.
4502*
TURNBULL, R.B., JR.
5171*
TUTELIAN, V.A.
3797, 4041
TUZHILIN, S.A.
4761
TYAGI, K.P.
4375*
TYLEN, U.
4738*
TYOCWA, M.
4851
UCHAIKIN, V.F.
4890
UCHVATKIN, V.G.
4447
UEDA, K.
5105
UEDA, M.
4061
UGOLEV, A.M.
4597
UHRICH, G.I.
4440
UJIKI, G.T.
4675
UNGENEUER, E.
4962
UNGER, J.L.
4701
UNGER, L.
4343
UPYREV, A.V.
4809
URAZOV, K.B.
5010
URBINA, A.M.
4734
URYVAEVA, P.V.
4144
USANOV, A.P.
4559
USBECK, W.
4404
USTIMENKO, Y.M.
4979
UTILI, R.
4879
VAGNE, M.
3926*
VAHOUNY, G.V.
3974*
VAILLANT, J.M.
5063
VAILLANT, R.
4094
VAILLE, C.
3979*, 3980*
VAISSADE, P.
3814*
VAJRABUKKA, T.
4835
VAKIL, B.J.
4507*
VAKSHURI, P.
4732
VALENKEVICH, L.N.
4615, 5099

VALERI, P.
4120
VALET, J.P.
4877, 4878
VALIGURA, T.A.S.
4575
VALLI, C.
4828
VAN CAMP, F.
4370*
VAN CANGH, P.J.
4938
VAN DER LINDEN, W.
4944*
VAN DER ZYPEN, E.
4489*
VAN HEERDEN, J.A.
4728
VAN HEIJENOORT, Y.
4167*
VAN HUSEN, N.
4222, 4262
VAN PUTTEN, W.J.
4681
VAN TONGEREN, J.H.M.
4216
VANAMEE, P.
4384*
VANECKOVA, O.
4322
VANNINI, V.
4062
VARADY, J.
4374*
VARDIMAN, J.W.
4648*
VARDY, P.A.
4544*
VARELA-DIAZ, V.M.
4193*
VARELA NINEZ, R.
4892
VARGA, B.
4166*
VARMA, R.R.
4784*
VARRO, V.
4205
VASILENKO, I.V.
4367*
VASILIEV, R.K.H.
4824
VASILIEVA, T.G.
5073
VASILYUK, V.N.
4889
VAUCLAIR, R.
4349
VAUGELADE, P.
3814*
VAYASSE, P.
4470
VECCHI, G.
4062
VECCHIONI, R.
4357
VEDRENNE, J.
4785*
VENABLES, C.W.
3922*
VENGRZHANOVSKII, P.N.
3827
VENKATADRI, G.
4983
VENNES, J.A.
4294
VENTZ, M.
4964
VERDONK, G.
4869
VERMA, S.P.
5154
VERMESSE, G.
4592
VERMGO, N.H.
5032
VERNER, J.V.
5121
VESSAL, K.
4195*
VETTER, D.
4395
VEYRIE, J.
4470
VEZERIDIS, M.
4168*
VICKSTROM, D.
4795*
VIDAL SANS, J.
5042
VIELLE, G.
4625
VIETCH, R.L.
4012*
VIIKARI, S.J.
4353
VILLAKO, K.
4481
VILLAR, H.V.
5093
VILLASMIL SALES, J.A.
4811
VINIK, A.
3958
VINIK, A.I.
3942*
VINOGRADOV, V.V.
4983
VISAKORPI, J.K.
4603*
VISHNEEVSKII, A.M.
4860*
VISKUM, K.
4533
VIVOLI, G.
4062
VIZI, E.S.
3890
VOEGELI, E.
5187
VOGEL, C.L.
4794*
VOGEL, F.S.
3804
VOGT, T.R.
4862*
VOIGT, J.
4571
VOIGT, T.
4148
VOINOVA, M.K.H.
4492*
VOIROL, M.
5059
VOLCHEK, V.M.
4560
VOLDMAN, C.
4402
VOLK, B.
4265
VOLKHEIMER, G.
3813*
VOLKOSLAVSKIA, N.V.V.
4801
VOLKOV, D.M.
3838
VOLLMAN, R.W.
4497*
VON BERGER, L.
4226
VON BERGMANN, K.
3994*, 5009
VON BRAND, T.
5152
VON BUREN, U.S.
4339
VON DER OELSNITZ, G.
4330
VON EKESPARRE, W.
4335
VON SCHENCK, H.
5160*
VON SEEBACH, N.B.
4848
VONDRASEK, P.
3880
VONMIKULICZ-RADECKI, J.G.
4443
VOROBIEVA, A.A.
4368*
VOTTELER, T.
4365
VOYER, M.
4855
WAGLE, S.R.
4051
WAGNER, H.N. JR.
4940*
WAGNER, R.
4825
WAGNER, V.P.
5137
WAITE, D.W.
4929
WAKABAYASHI, N.
4401
WAKEFIELD, T.
4047
WALKER, B.E.
4508*, 4525
WALKER, R.I.
4180
WALKER-SMITH, J.
4621

WALLAT, H.
4304
WALLAT, H.J.
4306
WALSCHMIDT, J.
4101
WALSH, J.H.
3936*, 4283, 4488*, 4491*
WALTER, J.F.
4790*
WANDS, J.R.
4871*
WANGER, F.
5187
WARNIR, R.A.
5191
WARD, A.S.
4490*
WARE, A.J.
4786*, 4875*
WARNER, G.T.
5161*
WARNES, T.W.
4904*
WARSHAW, A.L.
4197*
WARTER, J.
5070
WARWICK, F.
4284
WATANABE, A.
4014*
WATERFIELD, A.A.
3891
WATSON, A.J.
4635
WATSON, C.J.
4174
WATSON, L.
4904*
WATTINE, M.
3984
WAY, L.W.
4331
WAYNE, E.R.
5084
WEAKLEY, F.L.
5194
WEBB, J.N.
4310
WEBB, J.P.W.
4605*
WEBSTER, I.
4176
WEBSTER, P.D.
5131*
WEBSTER, P.D. III
3982*
WEE, K.P.
4758
WEETMAN, D.F.
3907
WEIA, H.D.
4253
WEIMANN, S.
4341
WEIMER, M.
4174
WEISBRODT, N.W.
3867*, 3868*
WEISEL, H.
4992
WEISER, P.
4647*
WEISS, R.
4385*
WEISS, W.
4821
WELCH, T.P.
5086
WELLERS, G.
3810*
WEMMER, U.
4588
WENZ, W.
4271
WENZE, W.
4212
WENZEL, F.J.
4949*
WENZL, M.
4963
WEPIERRE, J.
3918
WERMER, P.
5116
WERNER, V.R.
4566
WESOLOWSKI, H.
3915
WHARTON, B.A.
4642
WHITE, C.B.
4182
WIATROWSKI, E.
3855
WIELAND, O.H.
4095
WIENBECK, M.
4217, 4316
WIESENER, J.
4539
WILDGRUBE, H.J.
4125
WILK, P.J.
4703
WILKIE, R.C.
4729
WILKINSON, G.R.
4793*
WILL, J.A.
3920*
WILLEMS, G.
3964
WILLGERODT, H.
4608*
WILLIAMS, D.
4328
WILLIAMS, L.F.
4497*
WILLIAMS, R.
4901*
WILLIAMSON, J.T.
4050
WILLMAN V.L.
3977*
WILLMEN, H.R.
4668
WILLVONSEDER, R.
4782*
WILSON, D.E.
4862*
WILSON, J.
4377*
WILSON, W.F.
4729
WINAWER, S.J.
4384*, 4688
WINCKLER, K.
4419
WINDORFER, A., JR.
4854*
WING, R.M.
3853
WINGATE, D.L.
3831
WINKLER, R.
4405
WINTERER, J.C.
5096
WISE, L.
4644, 4952*
WISHER, M.H.
3794*
WISNIEWSKI, S.
4690
WISSINK, H.
4275
WITTE, O.
5097
WITTMAN, I.
4351
WOBSER, E.
4255
WOLFE, B.M.
4593
WOLFE, M.
3932*
WOLNICK, S.
4968
WOLPERS, C.
4998
WOOD, C.B.
4835
WOOD, J.D.
3892
WOOD, L.
4694
WOODLINGS, T.L.
4690
WOODWARD, E.R.
4001*
WORMSLEY, K.G.
4483*, 4502*
WRIGHT, L.F.
4509*
WRIGHT, M.G.
3917
WRIGHT, N.A.
4635
WRYZYKOWSKI, R.
4506*
WU, A.
4913*

WU, W.C.
4666
WUNSCH, E.
3887
YAJKO, R.D.
5085
YAKIMETS, W.W.
4707
YAKOB, L.E.
4890
YAKOVLEV, A.Y.
4146
YAKOVLEV, V.A.
4075
YAMAGISHI, T.
4459
YAMAGUCHI, T.
3871*
YAMAMOTO, M.
5074
YAMASAKI, M.
4706
YAMAZAKI, M.
4411
YARZABAL, L.A.
4193*
YEOMAN, L.C.
4019
YESUDOSS, S.
4172
YEVDOKIMOVA, G.S.
4075
YEVTIKHIEVA, E.P.
4445
YEVTIKHIVEA, E.P.
4445
YOEMANS, N.D.
3788*
YOSHIZAWA, S.
4162
YOSIDA, M.
5101
YOUNG, J.
4663
YOUNG, J.M.
3894
YOUNG, S.
5178
YOUNG, V.R.
5096
YUHAI, N.V.
4063
YULDASHEV, A.Y.
3801
YUM, K.Y.
4236
ZACCHI, T.
4896
ZAGNOLI, P.
4724
ZAHORODNEVA, A.H.
3959
ZAJEC, S.
3861*
ZALTSMANE, V.K.
4799
ZAMORA, C.S.
3920*
ZANNINI, G.
4836
ZANON, V.
5042
ZAPRUDNOV, A.M.
4201
ZARINIA, L.A.
4799
ZAVERI, M.P.
4504*
ZELDIN, V.E.
4860*
ZENGTELER, G.
4908
ZER, M.
4372*
ZERVOS, S.
4669
ZHELTVAI, V.V.
3960
ZHIGZHINSUREN, L.
4808
ZHURAVLEV
4810
ZHURAVLEV, V.A.
4270
ZHURAVLEVA, L.V.
5033
ZIEGLER, H.
4661
ZIEGLER, J.L.
4794*
ZIEGLER, R.
3953, 3954, 4494*
ZIEMLANSKI, S.
3820, 3824, 4153*
ZIEROTT, G.
4665
ZIGANSHIN, R.V.
5011
ZIKE, W.L.
4301, 4327, 4788*
ZIMMERMAN, X.B.
4933
ZIMMERMANN, H.B.
4939
ZINNEMAN, H.H.
4917
ZLATKINA, A.R.
4597
ZOLIN, V.P.
4560
ZOLOTAREVSKII, V.B.
4368*
ZSELI, J.
3890
ZUCCHETTI, F.
4895
ZUCKERMAN, A.J.
4901*
ZUFAROV, K.A.
3801, 4450
ZUIDEMA, G.D.
4739*
ZVOLANKOVA, K.
4754
ZYDOWICZ, L.
4930

NUMBERS FOLLOWED BY AN ASTERISK INDICATE ABSTRACTS. OTHER NUMBERS REFER TO CITATIONS.

ABDOMEN
  COMPLICATIONS
    PREVENTION, 5052
    SURGERY, 5052
  DRAINAGE
    CHOLECYSTECTOMY, 4972

ABDOMEN, ACUTE
  ETIOLOGY
    MECKELS DIVERTICULUM, 4575
  PERITONEOSCOPY, 4189*

ABSCESS
  ANUS
    CROHNS DISEASE, 5192
  ESOPHAGUS
    STENOSIS, 4336
  LIVER
    AMEBIASIS, 5137, 5142, 5146, 5154
    COMPLICATIONS, 5154
    DIAGNOSIS, 5142
    PREGNANCY, 5137
    THERAPY, 5142

ABSORPTION
  ALCOHOL
    CAFFEINE, 3836
  AMINO ACIDS
    GALLBLADDER, 3819*
    INTESTINE, 3810*
  BILE ACIDS
    GASTROINTESTINAL TRACT, 3859
    SHUNT, INTESTINAL, 4952*
  CALCIUM
    DIETARY FACTORS, 3845
    FEEDING, 4619
    OBESITY, 4619
    VITAMIN D, 3847
  CARBOHYDRATE
    NEONATE, 4608*
    PREMATURITY, 4608*
    SMALL INTESTINE, 3829
  CIRRHOSIS
    XYLOSE, 4930
  COLON
    REVIEWS, 3840
  DIETARY FACTORS, 3806*
  DUODENUM
    FATTY ACIDS, 3791*
    ULTRASTRUCTURE, 3791*
  ELECTROLYTE
    GASTRIN, 3839
    GLUCAGON, 3815*
    INTESTINE, 3830
    JEJUNUM, 3815*
  FATTY ACIDS, 3824
    INTESTINE, 3850
  FERRITIN, 3806*
  FOOD
    FUNGICIDES, 3846
    TRACE ELEMENT, 3846
  GASTROINTESTINAL
    HORMONE EFFECTS ON, 3877
    MORPHOLOGY, 3820
  GLUCOSE
    CELIAC DISEASE, 4605*
    DIETARY FACTORS, 4153*
    GASTRIN, 3839

ABSORPTION (continued)
  GLUCOSE (continued)
    GLUCAGON, 3815*
    JEJUNUM, 3815*
    SMALL INTESTINE, 3839
  ILEUM
    CELIAC DISEASE, 4605*
  INTESTINE
    DRUG EFFECTS ON, 3833
    FOOD ADDITIVES, 3917
    HEMODIALYSIS, 4190*
    MORPHOLOGY, 3800
    NERVOUS SYSTEM, 3827
    XYLOSE, 4231
  IRON
    DUODENUM, 3851
    FRUCTOSE, 5091
    INTESTINE, 3828
    NEONATE, 3828
    PANCREATECTOMY, 5059
  JEJUNUM
    HORMONE EFFECTS ON, 3808*
  LIPIDS
    BILE ACIDS, 3859
  OXALATE
    GASTROINTESTINAL DISEASE, 5026
  PROTEIN
    AGE FACTORS, 3832
    SMALL INTESTINE, 3832
  SMALL INTESTINE
    AGE FACTORS, 4615
    ALCOHOL, 3812*
    CELIAC DISEASE, 4637
    CHOLINESTERASE, 4158
    ENTEROCOLITIS, 4597
    FATTY ACIDS, 3849
    MONOAMINOXIDASE, 4158
    PATHOLOGY, 4637
    RADIOISOTOPES, 3838
    REVIEWS, 3826
  STOMACH
    ASPIRIN, 4391*
    BIOPOTENTIALS, 3843
  SUGAR, 3814
    GALLBLADDER, 3819*
  SULFATE
    ILEUM, 3848
  SURFACTANTS
    INTESTINE, 3823
  TRIGLYCERIDE
    BILE ACIDS, 3859
  VITAMIN B1
    DEFICIENCY, 3810*
  VITAMIN B2
    DEFICIENCY, 3810*
  VITAMIN B12
    ALCOHOL, 3812*
    SMALL INTESTINE, 3812*
  VITAMIN D
    KIDNEY DISEASE, 5067
  WATER
    CELIAC DISEASE, 4605*
    GASTRIN, 3839
    JEJUNUM, 3815*

ACETYLCHOLINE
  BINDING
    SMALL INTESTINE, 3894

ACETYCHOLINE (continued)
ILEUM
MOTILITY, 3870*

ACHALASIA
DIAGNOSIS, 4361
FAMILIAL FACTORS, 4321
SURGERY, 4322

ACHLORHYDRIA
SEE ALSO ACID SECRETION
PEPTIC ULCER
RECURRENCE, 4509*

ACID PHOSPHATASE
SEE PHOSPHATASE, ACID

ACID SECRETION
SEE ALSO ACHLORHYDRIA
SEE ALSO HYPERCHLORHYDRIA
SEE ALSO HYPOCHLORHYDRIA
HYPERCHLORHYDRIA
CALCIUM, 4507*
HISTAMINE, 4507*
PENTOSE
OXIDATION, 3950
PEPTIC ULCER
ANTICHOLINERGIC AGENTS, 4491*
CALCIUM, 4507*
ETHNIC FACTORS, 4504*
H2 RECEPTOR ANTAGONISTS, 4491*
HISTAMINE, 4504*, 4507*
STOMACH
ATROPINE, 3965, 4499*
CHILDREN, 4215
CHOLINERGIC AGENTS, 3956
DIETARY FACTORS, 3931*
DRUG EFFECTS ON, 3972, 4519
ENZYMES, 3947
FATTY ACIDS, 3930*
GASTRIN, 3956, 3973
H2 RECEPTOR ANTAGONISTS, 3972
HEIDENHAIN POUCH, 3946*
HISTAMINE, 3972, 4492*
HORMONE CONTROL, 3944*, 3945*, 3951, 3954, 4396
HYPNOSIS, 3941*
INTESTINE, 3946*
ION TRANSPORT, 3949
NERVOUS CONTROL, 4396
PEPTIC ULCER, 4488*, 4492*, 4503*, 4505*, 4511*, 4519
PROSTAGLANDIN, 3927*
TECHNIQUES, 4215

ACIDITY
ANTRUM
GASTRIN, 3966
ESOPHAGUS
PHYSICAL FACTORS, 3862*
STOMACH
ANTACIDS, 4414
ENDOSCOPY, 4397
PATHOLOGY, 4386*
PERMEABILITY, 3844
PHYSICAL FACTORS, 3862*
TECHNIQUES, 4397

ACIDS
ESOPHAGUS
PERMEABILITY, 4301*
STOMACH
MOTILITY, 3889

ACRODERMATITIS ENTEROPATHICA
COPPER, 5128
DRUG TREATMENT
ZINC, 5113
ZINC, 5128
DEFICIENCY, 5129

ADENOSINE TRIPHOSPHATE
CECUM
MOTILITY, 3907

ADENYL CYCLASE
JEJUNUM
HORMONE EFFECTS ON, 3817*
LIVER
ULTRASTRUCTURE, 3794*
STOMACH
FEEDING, 3970
IRRADIATION, 3970

ADHESIVES
SEE TISSUE ADHESIVES

AFLATOXIN
HEPATITIS, NONVIRAL, 4861*

AGE FACTORS
SEE ALSO CHILDREN, NEONATE
CHOLECYSTITIS
COMPLICATIONS, 4976
SURGERY, 4981
CHOLELITHIASIS
COMPLICATIONS, 4969
EPIDEMIOLOGY, 4960
SURGERY, 4969
GASTROINTESTINAL TRACT
IRRADIATION, 4164*
INDOCYANINE GREEN
BINDING, 3998*
LIVER
CORTICOSTEROIDS, 4096
CYTOCHROMES, 4083
LIVER FUNCTION TESTS
ATHEROSCLEROSIS, 4842
HYPERTENSION, 4842
MALABSORPTION
DIAGNOSIS, 4616
THERAPY, 4616
PANCREAS
SECRETION, 4773
PANCREATITIS
EPIDEMIOLOGY, 4778
PEPTIC ULCER, 4485*
DRUG-INDUCED, 4538
PROTEIN
ABSORPTION, 3832
SYNTHESIS, 5096
SALIVARY GLANDS
ENZYMES, 3913
SMALL INTESTINE
ABSORPTION, 4615
ENZYMES, 4615

AGE FACTORS (continued)
STOMACH
ATROPHY, 4373*, 4393
NEOPLASMS MALIGNANT, 4393
NEOPLASMS, MALIGNANT, 4453, 4460
ULCER, 4393

ALBUMIN
BILIRUBIN
TRANSPORT, 4123
HYPERBILIRUBINEMIA, 4855
METABOLISM
LIVER, 4092
SYNTHESIS
INFLAMMATION, 4085
LIVER, 4085

ALCOHOL
ABSORPTION
CAFFEINE, 3836
AMMONIA
METABOLISM, 4050
FATTY ACIDS
OXIDATION, 4154*
LIPIDS
METABOLISM, 4151*, 4154*
LIVER
DRUG METABOLISM, 4010*
MALABSORPTION
DRUG-INDUCED, 4641
OXIDATION
CATALASE, 3997*
LIVER, 3996*, 3997*, 4049
PANCREAS
METABOLISM, 3982
SECRETION, 3982, 4767
PANCREAS DISEASE
REVIEWS, 4767
PANCREATITIS
THERAPY, 4764
PANCREATITIS, CHRONIC
CALCIFICATION, 4765
PEROXIDATION
LIVER, 4002*
SMALL INTESTINE
ABSORPTION, 3812*
DISACCHARIDASE, 4151*
DRUG ABSORPTION, 3858
STOMACH
BLEEDING, 4424
PERMEABILITY, 3938*
UREA
SYNTHESIS, 4050
VITAMIN B6
METABOLISM, 4012*
VITAMIN B12
ABSORPTION, 3812*

ALCOHOL DEHYDROGENASE
BLOOD
LIVER DISEASE, 4812

ALCOHOLIC LIVER DISEASE
SEE LIVER DISEASE, ALCOHOLIC

ALCOHOLISM
CIRRHOSIS
AUTOIMMUNITY, 4917

ALCOHOLISM (continued)
ESOPHAGUS
NEOPLASMS, MALIGNANT, 5094
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 5094
INTESTINE
MALABSORPTION, 3835
LIVER
NEOPLASMS, MALIGNANT, 5094
PANCREAS
NEOPLASMS, MALIGNANT, 5094
SECRETION, 4766
PANCREAS DISEASE, 4723*
REVIEWS, 4730
PANCREATITIS
ETIOLOGY, 4777
STOMACH
NEOPLASMS, MALIGNANT, 5094

ALCOHOLS, POLYHYDRIC
SMALL INTESTINE
DISACCHARIDE, 4162

ALKALINE PHOSPHATASE
SEE PHOSPHATASE, ALKALINE

ALKALOIDS
SMALL INTESTINE
CIRCADIAN RHYTHM, 4156
KINETICS, CELL, 4156

AMEBIASIS
ABSCESS
LIVER, 5146
DIAGNOSIS
TECHNIQUES, 4242
DRUG TREATMENT, 5147
INFLAMMATORY BOWEL DISEASE
COLITIS, 5131*
LARGE INTESTINE
ETIOLOGY, 5156
SURGERY, 5155
LIVER
ABSCESS, 5137, 5142, 5154
DIAGNOSIS, 5149
DRUG TREATMENT, 5149

AMIDES
LIVER INJURY
PREVENTION, 4107

AMINES
METABOLISM
LIVER, 4072
STARVATION, 4072
PANCREAS
SECRETION, 3979*
PEPTIC ULCER
SIMULATION, 4501*

AMINO ACIDS
ABSORPTION
GALLBLADDER, 3819*
INTESTINE, 3810*
BLOOD
SECRETIN, 3937*
GASTROENTERITIS
NEONATE, 5087

AMINO ACIDS (continued)
LIVER
REGENERATION, 4017
LIVER INJURY
PREVENTION, 4107
METABOLISM
BILIARY DISEASE, 4975
CIRCULATION, 3811*, 4852
COENZYMES, 4091
GALLBLADDER DISEASE, 4975
LIVER, 3811*, 4078, 4090, 4091
PENTAGASTRIN, 3933*
STOMACH, 3933*
VITAMIN A, 4090
VITAMIN B6, 4091
VITAMIN E, 4078
STOMACH
ASPIRIN, 4391*
TRANSPORT
BILE ACIDS, 3837

AMMONIA
METABOLISM
ALCOHOL, 4050
TOLERANCE TEST
LIVER, 4101
TRANSPORT
COLON, 4654*

AMPULLA OF VATER
STENOSIS
SIMULATION, 4139

AMYLASE
BILE DUCT
OBSTRUCTION, 4197*
GALLBLADDER
BILE, 4965
PANCREATITIS
DIAGNOSIS, 4197*
SECRETION
SALIVARY GLANDS, 3911

ANALGESICS
DUODENUM
RADIOLOGY, 4411
STOMACH
MOTILITY, 4377*
RADIOLOGY, 4411

ANEMIA
CELIAC DISEASE
REVIEWS, 4634
CIRRHOSIS, 4926
CROHNS DISEASE, 5172*
COLON, 5179
DRUG TREATMENT, 5195
SMALL INTESTINE
NEOPLASMS, BENIGN, 4564
NEOPLASMS, MALIGNANT, 4601
STOMACH
PATHOLOGY, 4380*

ANEMIA, SICKLE CELL
LIVER
BIOPSY, 4840

ANGIOGRAPHY
BILE DUCT
SURGERY, 5012
BILIARY
CONTRAST MEDIA, 4279
STENOSIS, 4259
BILIARY DISEASE
ENDOSCOPY, 4288

ANESTHETICS
HEPATITIS, NONVIRAL
DRUG-INDUCED, 4863
LIVER INJURY
DRUG-INDUCED, 4857*
TECHNIQUES, 4281
BILIARY TRACT
COMPLICATIONS, 4259
CHOLELITHIASIS, 5000
COLON
POLYPS, 4677
GALLBLADDER DISEASE, 4276
ENDOSCOPY, 4287
TECHNIQUES, 4287
GASTROINTESTINAL TRACT
BLEEDING, 5022
HEPATITIS, VIRAL
CIRRHOSIS, 4881
JAUNDICE, OBSTRUCTIVE, 4789*
LIVER
CONTRAST MEDIA, 4106
CYSTS, 4869
LYMPHATICS, 4817
NEOPLASMS, BENIGN, 4825
PORTACAVAL SHUNT, 4836
LIVER DISEASE, 4270, 4810
PATHOLOGY, 4294
MECKELS DIVERTICULUM
BLEEDING, 4545*
PANCREAS
NEOPLASMS, MALIGNANT, 4724

ANGIOTENSIN
JEJUNUM
BIOPOTENTIALS, 3884
MOTILITY, 3884
STOMACH
BIOPOTENTIALS, 3884
MOTILITY, 3884

ANOMALY
ESOPHAGUS
THERAPY, 4307
STOMACH
OBSTRUCTION, 4477

ANOMALY, CONGENITAL
BILIARY
DILATATION, 4961
CELIAC-AXIS COMPRESSION SYNDROME, 5070
ENTEROCOLITIS
SURGERY, 5173*
ESOPHAGUS
STENOSIS, 4358
GALLBLADDER, 5005
COMPLICATIONS, 4971
EPIDEMIOLOGY, 4971
MOTILITY, 4958
SURGERY, 4970, 4971

ANOMALY, CONGENITAL (continued)
PANCREAS
SURGERY, 4725
PYLORUS
STENOSIS, 4469
SHORT BOWEL SYNDROME
FAMILIAL FACTORS, 4547
SMALL INTESTINE
BLEEDING, 4235
DIAGNOSIS, 4235
FAMILIAL FACTORS, 4547
STOMACH
PYLORUS, 4480

ANORECTUM
NEOPLASMS, MALIGNANT
COMPLICATIONS, 4697
RADIOLOGY, 4696
SURGERY, 4697
PRESSURE STUDIES
INCONTINENCE, 4651*
MEGACOLON, 4651*
STRICTURE
CROHNS DISEASE, 5163*

ANTACIDS
ESOPHAGITIS, REFLUX, 4316
ESOPHAGUS DISEASE
DRUG TREATMENT, 4302
PEPTIC ULCER, 4520
STOMACH
ACIDITY, 4414
STOMACH DISEASE
DRUG TREATMENT, 4302

ANTIBACTERIALS
GASTROINTESTINAL
ENZYMES, 3991
HEPATITIS, NONVIRAL
DRUG-INDUCED, 4858*, 4859*, 4864
JAUNDICE
ETIOLOGY, 4846
LIVER COMA
DRUG-INDUCED, 4865
LIVER INJURY
DRUG-INDUCED, 4867
PANCREAS
ENZYMES, 3991

ANTIBIOTICS
SEE ALSO ANTIBACTERIALS, DRUG TREATMENT
BILIARY
INFECTION, BACTERIA, 4986
COLITIS
DRUG INDUCED, 4655*, 4701, 4702
DIARRHEA
DRUG-INDUCED, 5013*
EXCRETION
BILE, 4120
GASTROENTERITIS
CHILDREN, 5092
GASTROINTESTINAL
ENZYMES, 3991
HYPERBILIRUBINEMIA, 4855
INTESTINE
MOTILITY, 5013*
LIVER
DRUG METABOLISM, 4010*

ANTIBIOTICS (continued)
LIVER COMA
DRUG-INDUCED, 4865
LIVER DISEASE
COMPLICATIONS, 4798
METABOLISM
BILIARY DISEASE, 4798
LIVER DISEASE, 4798
MICROORGANISMS
SIMULATION, 4183
PANCREAS
ENZYMES, 3991
SECRETION, 3983
PANCREAS FUNCTION TESTS
DRUG EFFECTS ON, 4772
PANCREATITIS
DRUG TREATMENT, 4739*
SMALL INTESTINE
MICROORGANISMS, 4171
ZINC
METABOLISM, 4069

ANTICHOLINERGIC AGENTS
PEPTIC ULCER, 4520
ACID SECRETION, 4491*
GASTRIN, 4491*
STOMACH
SECRETION, 3957
ULCERATIVE COLITIS, 4711*

ANTICOAGULANTS
LARGE INTESTINE
OBSTRUCTION, 4669

ANTIDEPRESSANTS
BILE
EXCRETION, 4138
METABOLISM
CHOLESTASIS, 4138

ANTIEMETICS
PYLORUS
OBSTRUCTION, 4372*

ANTIGEN
HEPATITIS, VIRAL
EPIDEMIOLOGY, 4878
RADIOIMMUNOASSAY, 4878
PARASITES AND PARASITIC DISEASE
ELECTROPHORESIS, 4193*

ANTIGEN, AUSTRALIA
CIRRHOSIS
COMPLEMENT, 4902*
EPIDEMIOLOGY, 4814
HEPATITIS, CHRONIC
IMMUNITY, 4901*
SMALL INTESTINE, 4897
HEPATITIS, VIRAL
CARRIER STATE, 4898
CHILDREN, 4876*
CORTICOSTEROIDS, 4870*, 4882
ELECTROPHORESIS, 4896
EPIDEMIOLOGY, 4877, 4882, 4893
FECES, 4896
HEPATITIS, CHRONIC, 4876*, 4879
IMMUNOLOGY, 4873*, 4880
RADIOIMMUNOASSAY, 4896

ANTIGEN, AUSTRALIA (continued)
HEPATITIS, VIRAL (continued)
SMALL INTESTINE, 4897
STATISTICAL STUDY, 4877, 4893
URINE, 4896
LIVER
NEOPLASMS, MALIGNANT, 4814, 4826
LIVER DISEASE
LIVER DISEASE, 4813
RADIOIMMUNOASSRY, 4813

ANTIGEN, CARCINOEMBRYONIC
COLON
NEOPLASMS, MALIGNANT, 4244, 4646*
INTESTINE
NEOPLASMS, MALIGNANT, 4178

ANTIHISTAMINES
PANCREATITIS
DRUG TREATMENT, 4756

ANTIINFLAMMATORY AGENTS
PEPTIC ULCER
DRUG-INDUCED, 4500*, 4538
STOMACH
DRUG EFFECTS ON, 4381*
PERMEABILITY, 3938*

ANTINEOPLASTIC AGENTS
DISACCHARIDE
MALABSORPTION, 4157
PANCREATITIS
DRUG-INDUCED, 4758
PREVENTION, 4740*
SMALL INTESTINE
DISACCHARIDASE, 4157
DRUG ABSORPTION, 3860

ANTISPASMODICS
SMALL INTESTINE
MOTILITY, 3896

ANTITRYPSIN, ALPHA
BILE DUCT
HYPSPLASIA, 4838
DEFICIENCY
CHOLESTASIS, 4838
CIRRHOSIS, 4934
CIRRHOSIS, BILIARY, 4838
GENETIC FACTORS, 4838

ANTRECTOMY
SEE ALSO GASTRECTOMY
SEE ALSO STOMACH, SURGERY
BLOOD
GASTRIN, 3924*
ESOPHAGUS
SPHINCTER, 4486*
PEPTIC ULCER, 4508*
VAGOTOMY, 4525
SEQUELAE
ENTERITIS, 4402
STOMACH
REGENERATION, 4400
SECRETION, 3928*, 4400

ANTRUM
SEE ALSO STOMACH
BLOOD
GASTRIN, 3962
GASTRIN
ACIDITY, 3966
CHEMICAL COMPOSITION, 3942*
NERVOUS CONTROL, 3936*
GASTRITIS, ATROPHIC
EPIDEMIOLOGY, 4491
ION TRANSPORT
REVIEWS, 3842
MOTILITY
VAGOTOMY, 3866*

ANUS
SEE ALSO LARGE INTESTINE
ABSCESS
CROHNS DISEASE, 5192
FISSURE
CROHNS DISEASE, 5194
FISTULAS
CROHNS DISEASE, 5192
MORPHOLOGY, 3795
FISTULA, 4674
NEOPLASMS, MALIGNANT
CHEMOTHERAPY, 4692
RADIOTHERAPY, 4692
SURGERY, 4692
ULCER
CROHNS DISEASE, 5192

APPENDICITIS
DIAGNOSIS, 4650*
DIETARY FACTORS
FIBER, 5089
THERAPY, 4650*

APPENDIX
SEE ALSO LARGE INTESTINE
NEOPLASMS, MALIGNANT, 4686
POLYPS, 4691
TUBERCULOSIS, 4705

ARTERY
EROSION
PANCREAS DISEASE, 4729

ASBESTOS
ENVIRONMENTAL FACTORS, 4176
FEEDING
PRIMATES, 4176
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 4176

ASCARIASIS
DRUG TREATMENT, 5143

ASCITES
CIRRHOSIS
ETIOLOGY, 4928
SURGERY, 4932
SURVIVAL, 4937
DIURETICS, 4919*
DRUG TREATMENT, 4931
PHOSPHOLIPASE
CIRRHOSIS, 4741*
PANCREATITIS, 4741*

ASPIRIN
GASTROINTESTINAL
BLEEDING, 4437, 4438
METABOLISM
HYPERBILIRUBINEMIA, 4854*
PHOTOTHERAPY, 4854*
STOMACH
ABSORPTION, 4391*
AMINO ACIDS, 4391*
BIOPOTENTIALS, 4376*
EROSIONS, 4388*, 4391*

ATHEROSCLEROSIS
LIVER FUNCTION TESTS
AGE FACTORS, 4842

ATRESIA
BILIARY
DIAGNOSIS, 4194*
ESOPHAGUS
SURGERY, 4320
SMALL INTESTINE
DIAGNOSIS, 4546
THERAPY, 4546

ATROPHY
HEPATITIS, VIRAL
PERFUSION, 4895
PANCREAS
MOTILITY, 3882
STOMACH
AGE FACTORS, 4373*, 4393
RADIOLOGY, 4373*

ATROPINE
BLOOD
GASTRIN, 3965
SMALL INTESTINE
MOTILITY, 3863*
STOMACH
ACID SECRETION, 3965, 4499*
ELECTROLYTE, 4499*
PEPSIN, 4499*

AUTOIMMUNITY
CIRRHOSIS
ALCOHOLISM, 4917
HEPATITIS, CHRONIC, 4796*
CIRRHOSIS, BILIARY, 4796*
ETIOLOGY, 4900*
HEPATITIS, VIRAL, 4796*
LIVER DISEASE
SCLERODERMA, 4904*

AUTORADIOGRAPHY
INTESTINE
KINETICS, CELL, 3802
LIVER
KINETICS, CELL, 3802
SMALL INTESTINE
KINETICS, CELL, 3801

BACTERIA
BILE ACIDS
METABOLISM, 4167*
COLON, 4169
SMALL INTESTINE
SPRUE, TROPICAL, 4604*

BARIUM
CELIAC DISEASE
RADIOLOGY, 4630
ENEMA
LAXATIVES, 4243
TECHNIQUES, 4243
STOMACH
BIOPOTENTIALS, 3923*
SECRETION, 3923*

BEHAVIOR
ULCER
STRESS, 3921*

BEZOARS
SEE FOREIGN BODIES

BICARBONATE
SECRETION
PANCREATITIS, 4754

BICARBONATE SECRETION
CHLORIDE
TRANSPORT, 4152*
SALIVARY GLANDS, 3825
SODIUM
TRANSPORT, 4152*

BILE
ANTIBIOTICS
EXCRETION, 4120
BILE ACIDS
CHOLELITHIASIS, 4950*
BILIRUBIN
EXCRETION, 4120
BLOOD
GASTRIN, 3988
CHEMICAL COMPOSITION
PORTACAVAL SHUNT, 4136
ENZYMES, 4003*
EXCRETION
ANTIDEPRESSANTS, 4138
CHOLESTASIS, 4138
PORTACAVAL SHUNT, 4136
GALLBLADDER
AMYLASE, 4965
GLUCOSE, 4993
LIPIDS
COENZYMES, 3975*
LIPASE, 3975*
SECRETION, 4941*
PANCREAS
SECRETION, 3985
PERITONITIS, 5042
PHOSPHATASE, ALKALINE
BLOOD, 4004*
PIGMENTS
LIVER DISEASE, 4103
REFLUX
EROSIONS, 3939*
ULCER, 3929*
SECRETION
BILIARY DISEASE, 4806
CHOLERETIC AGENTS, 4806
DRUG EFFECTS ON, 4967
LIVER DISEASE, 4806
NARCOTICS, 4132
VAGOTOMY, 4967

BILE (continued)
STRESS
PEPTIC ULCER, 4539
SULFOBROMOPHTHALEIN
EXCRETION, 4120
SYNTHESIS
INFECTION, BACTERIA, 4137
PHENOBARBITAL, 3994*
STEROID, 3994*

BILE ACIDS
ABSORPTION
GASTROINTESTINAL TRACT, 3859
SHUNT, INTESTINAL, 4952*
AMINO ACIDS
TRANSPORT, 3837
BILE
CHOLELITHIASIS, 4950*
BLOOD
JAUNDICE OBSTRUCTIVE, 4130
CHOLESTASIS
SIMULATION, 4000*
CHOLESTEROL
SECRETION, 4009*
ESOPHAGITIS
ETIOLOGY, 4301*
ESOPHAGUS
PERMEABILITY, 4301*
EXCRETION
CHOLECYSTECTOMY, 4133
HIRSCHSPRUNGS DISEASE, 4656*
INTESTINE
JAUNDICE OBSTRUCTIVE, 4130
LIPIDS
ABSORPTION, 3859
METABOLISM
BACTERIA, 4167*
CHOLECYSTECTOMY, 4133
CHOLELITHIASIS, 4125, 5002, 5009
CHOLESTEROL, 4125
GALLBLADDER DISEASE, 4125
GNOTOBIOSIS, 4167*
LIVER, 3807*
MALABSORPTION, 4617
SPECIES DIFFERENCES, 4013*
PEPSIN
SECRETION, 3935*
PEPTIC ULCER
SIMULATION, 4539
STOMACH
BIOPOTENTIALS, 4376*
PATHOLOGY, 4386*
PEPSIN, 3935*
PERMEABILITY, 3938*
STRESS
PEPTIC ULCER, 4539
SUGAR
TRANSPORT, 3837
SYNTHESIS
CHOLESTEROL, 4129
DIETARY FACTORS, 4129
LIVER, 4128, 4129
NARCOTICS, 4132
TRIGLYCERIDE
ABSORPTION, 3859

BILE DUCT
CHOLECYSTECTOMY
SEQUELAE, 4956
COMPRESSION
LYMPHATICS, 5008
CYSTS, 4956
DRAINAGE, 4985
PERMEABILITY, 4973
FISTULA
GALLBLADDER DISEASE, 5006
HYPOPLASIA
ANTITRYPSIN, ALPHA, 4838
INFLAMMATION
DIAGNOSIS, 4974
KINETICS, CELL
CARBON TETRACHLORIDE, 4060
LIVER
LIVER DISEASE, 4799
MORPHOLOGY, 4126
MORPHOLOGY
NERVOUS SYSTEM, 4127
NEOPLASMS
DRAINAGE, 4977
NEOPLASMS, MALIGNANT
CHEMOTHERAPY, 4947*
RADIOTHERAPY, 4947*
SURGERY, 4947*, 5007
OBSTRUCTION
AMYLASE, 4197*
BIOPSY, 4948*
DIAGNOSIS, 4197*
ECHINOCOCCOSIS, 5139
SURGERY, 4990
UROGENITAL SYSTEM, 4006*
PATHOLOGY
REVIEWS, 5068
PERFUSION
PRESSURE STUDIES, 4973
RADIOLOGY, 4277
CHOLECYSTITIS, 4940*
STRICTURE
CHOLEDOCHOLITHIASIS, 4988
PANCREATITIS, 4988
SURGERY, 4946*
SURGERY
ANGIOGRAPHY, 5012
PRESSURE STUDIES, 5012
RECURRENCE, 4959
REVIEWS, 5068

BILE SALTS
SEE BILE ACIDS

BILIARY
ANGIOGRAPHY
CONTRAST MEDIA, 4279
ATRESIA
DIAGNOSIS, 4194*
DILATATION
ANOMALY, CONGENITAL, 4961
DRAINAGE
CHOLECYSTITIS, 4984
COMPLICATIONS, 4983
TECHNIQUES, 4983, 4984
EXCRETION
HORMONE, 3852
STEROID, 3857

BILIARY (continued)
INFECTION, BACTERIA
ANTIBIOTICS, 4986
RADIOLOGY
TECHNIQUES, 4273
STENOSIS
ANGIOGRAPHY, 4259
SURGERY
CHELATING AGENTS, 4995
TRAUMA
DIAGNOSIS, 4966
SURGERY, 4966

BILIARY DISEASE
AMINO ACIDS
METABOLISM, 4975
ANGIOGRAPHY
TECHNIQUES, 4281
ANTIBIOTICS
METABOLISM, 4798
BILE
SECRETION, 4806
BLOOD
CLOTTING, 4980
DIAGNOSIS, 4278, 4809
ENDOSCOPY, 4256, 4257, 4272
ANGIOGRAPHY, 4288
GALLBLADDER
RADIOLOGY, 4992
LAPAROSCOPY, 4808
PORTAL HYPERTENSION
DISEASES ASSOCIATED WITH, 4957
RADIOLOGY, 4250
SCANNING, SCINTILLATION, 4250

BILIARY TRACT
ANGIOGRAPHY
COMPLICATIONS, 4259
NUCLEIC ACIDS
CORTICOSTEROIDS, 4131
SURGERY
PORTAL HYPERTENSION, 4957

BILIRUBIN
BINDING
ERYTHROCYTE, 4122
MEMBRANE, 4122
PROTEIN, 4181
EXCRETION
BILE, 4120
GILBERTS DISEASE
DIAGNOSIS, 4191*
LIVER INJURY
CARBON TETRACHLORIDE, 4124
METABOLISM
GILBERTS DISEASE, 4191*
HYPERBILIRUBINEMIA, 4854*
PHENOBARBITAL, 4124
PHOSPHOLIPID, 4119
PHOTOTHERAPY, 4854*
REVIEWS, 4116, 4174
PROTEIN
BINDING, 4118
SYNTHESIS
ENZYMES, 4005*
TRANSPORT
ALBUMIN, 4123
DRUG EFFECTS ON, 4123
ENZYMES, 4121

BILIRUBIN (continued)
TRANSPORT (continued)
LIVER, 4117, 4119, 4121, 4123
PHOSPHOLIPID, 4119

BIOCHEMISTRY
HELMINTHIASIS, 5152

BIOPOTENTIALS
COLON
MOTILITY, 3904
DUODENUM
HORMONES, 3831
MOTILITY, 3901
VAGOTOMY, 3883
GASTROINTESTINAL
MOTILITY, 3878
TECHNIQUES, 5019*
JEJUNUM
ANGIOTENSIN, 3884
ODDI, SPHINCTER OF
DRUG EFFECTS ON, 3865*
RECTUM
HYPERTENSION, 4663
SMALL INTESTINE
VAGOTOMY, 3868*
STOMACH
ABSORPTION, 3843
ANGIOTENSIN, 3884
ASPIRIN, 4376*
BARIUM, 3923*
BILE ACIDS, 4376*
ISCHEMIA, 3861*
VAGOTOMY, 3883, 4530

BLEEDING
CIRRHOSIS
COMPLICATIONS, 4927
COLON
CROHNS DISEASE, 5179
ENDOSCOPY, 4666
DUODENITIS
EROSIONS, 4426
DUODENUM
SURGERY, 5085
ESOPHAGUS
CIRRHOSIS, 4932, 4938
SURGERY, 4308, 5085
THERAPY, 4184
VARICES, 4184, 4308, 4938
GASTRITIS, EROSIVE, 4426
EPIDEMIOLOGY, 4382*
SURGERY, 4379*
GASTROINTESTINAL, 5036
ASPIRIN, 4437, 4438
CIRRHOSIS, 4264
CROHNS DISEASE, 5187
DIAGNOSIS, 5081
DRUG-INDUCED, 4374*, 4421, 4437
DRUG TREATMENT, 5060
ENDOSCOPY, 4206, 4208, 4264, 5130
HEMATEMESIS, 5126
LEUKEMIA, 5029
MELENA, 5126
PANCREAS DISEASE, 4729
RADIOLOGY, 4207
SIMULATION, 4438
SURGERY, 5035, 5061, 5062

BLEEDING (continued)
GASTROINTESTINAL (continued)
TELANGIECTASIA, 5123
THERAPY, 5130
VASOPRESSIN, 5015*
GASTROINTESTINAL TRACT
ANGIOGRAPHY, 5022
HODGKINS DISEASE
DUODENUM, 4557
LARGE INTESTINE
DISEASES ASSOCIATED WITH, 4661
RADIOLOGY, 4212
SURGERY, 4661
LIVER
MORPHOLOGY, 4145
NECROSIS, 4937
LIVER DISEASE
THERAPY, 4804
TRANSFUSION, 4804
VASOPRESSIN, 4804
LIVER FUNCTION TESTS, 4145
MECKELS DIVERTICULUM
ANGIOGRAPHY, 4545*
NEOPLASMS, BENIGN, 4578
SCANNING, SCINTILLATION, 4545*
PANCREATITIS
DRUG TREATMENT, 4750
SURGERY, 4750, 4752
PEPTIC ULCER
COMPLICATIONS, 4535
RECTUM
RADIOLOGY, 4212
SCANNING, SCINTILLATION, 4210
RECURRENCE
CROHNS DISEASE, 5187
SMALL INTESTINE
ANOMALY, CONGENITAL, 4235
EROSIONS, 4425
RADIOLOGY, 4212
STOMACH
ALCOHOL, 4424
DRUG-INDUCED, 4374*, 4424
ELECTROPHYSIOLOGY, 3925*
EROSIONS, 4425
GASTRITIS, 4421
NEOPLASMS, MALIGNANT, 4461
PEPTIC ULCER, 4421
SIMULATION, 4438
SURGERY, 4421, 5085
THERAPY, 4184, 4420
ULCER, 4369*
VARICES, 4184
ULCER
CIRRHOSIS, 4927
SURGERY, 4478

BLIND LOOP SYNDROME
CROHNS DISEASE
COMPLICATIONS, 5195
SMALL INTESTINE
PATHOLOGY, 4628

BLOOD
ALCOHOL DEHYDROGENASE
LIVER DISEASE, 4812
BILE ACIDS
JAUNDICE, OBSTRUCTIVE, 4130
CHOLECYSTOKININ
CELIAC DISEASE, 4609*

BLOOD (continued)
CHOLESTEROL
DRUG EFFECTS ON, 5108
CLOTTING
BILIARY DISEASE, 4980
CHOLEDOCHOLITHIASIS, 4980
HEPATITIS, CHRONIC, 4899*
HEPATITIS, VIRAL, 4885, 4886, 4887
LIVER DISEASE, 4980
PANCREATITIS, 4753
FATTY ACIDS
PANCREATITIS, 4755
FIBRINOLYSIS
GASTRITIS, 4394
GASTRIN, 5059
ANTRECTOMY, 3924*
ANTRUM, 3962
ATROPINE, 3965
BILE, 3988
BLOOD GROUP, 3969
CHEMICAL COMPOSITION, 3942*, 3973
CIRCADIAN RHYTHM, 3932*
DIETARY FACTORS, 3931*
FEEDING, 3932*
H2 RECEPTOR ANTAGONISTS, 3958
HISTAMINE, 3958
HYPERCALCEMIA, 3943*
PEPTIC ULCER, 4488*
PROSTAGLANDIN, 3967
TECHNIQUES, 4226
VAGOTOMY, 3924*
HEPATITIS, CHRONIC
FIBRINOLYSIS, 4899*
HISTAMINE
HEPATITIS, VIRAL, 4891
LIVER, 4098
PANCREATITIS, 4756
IMMUNOGLOBULINS
HEPATITIS, CHRONIC, 4903*
HEPATITIS, VIRAL, 4874*
LIPASE
PANCREATITIS, CHRONIC, 4253
LIPIDS
CHOLELITHIASIS, 5001
PANCREATITIS, 4755
PHOSPHATASE, ALKALINE
BILE, 4004*
PHOSPHOLIPASE
PANCREATITIS, 4741*
PHOSPHOLIPID
CIRRHOSIS, 4923*
HEPATITIS, CHRONIC, 4923*
PLATELETS
LIVER DISEASE, 4803
PROTEIN
HEPATECTOMY, 4016
SECRETIN
AMINO ACIDS, 3937*
FATTY ACIDS, 3937*
PEPTIC ULCER, 4490*
SMOKING, 4490*
SUGAR, 3937*
SEROTONIN
PANCREATITIS, 4756
SODIUM
CIRRHOSIS, 4919*
DIURETICS, 4919*

BLOOD (continued)
VITAMIN A
CIRRHOSIS, 4262
CROHNS DISEASE, 4262
MALABSORPTION, 4262
VITAMIN B6
PEPTIC ULCER, 4482*
VITAMIN B12
HEPATITIS, VIRAL, 4883

BLOOD FLOW
SEE CIRCULATION

BLOOD GROUP
BLOOD
GASTRIN, 3969

BRADYKININ
COLON
MOTILITY, 3864*
STOMACH
MOTILITY, 3864*

BRUCELLOSIS
LIVER
INFECTION, BACTERIA, 4792*

BURNS
STRESS
ULCER, 4390*

CADMIUM
ANALYSIS
OCCUPATIONAL FACTORS, 4866
DRUG METABOLISM
LIVER, 4058
LIVER
OCCUPATIONAL FACTORS, 4866

CAFFEINE
ALCOHOL
ABSORPTION, 3836

CALCIFICATION
PANCREATITIS, CHRONIC
ALCOHOL, 4765
EPIDEMIOLOGY, 4742*
ETIOLOGY, 4742*
PATHOLOGY, 4737*

CALCIUM
ABSORPTION
DIETARY FACTORS, 3845
FEEDING, 4619
OBESITY, 4619
VITAMIN D, 3847
FLUORIDE
METABOLISM, 3855
GALLBLADDER
MOTILITY, 4736
HYPERCHLORHYDRIA
ACID SECRETION, 4507*
PANCREAS
ENZYMES, 4736
SECRETION, 3986
PEPTIC ULCER
ACID SECRETION, 4507*

CALCIUM (continued)
PROTEIN
BINDING, 4163
TRANSPORT
INTESTINE, 3853
VITAMIN D, 3853

CALCULI
GALLBLADDER
PATHOLOGY, 4998
LIVER DISEASE
SURGERY, 4847

CANCER
SEE NEOPLASMS, MALIGNANT

CARBENOXALONE
DUODENUM
ULCER, 4503*
PEPTIC ULCER
DRUG TREATMENT, 4370*
STOMACH
GLYCOPROTEINS, 4503*
ULCER, 4370*, 4503*

CARBOHYDRATE
SEE ALSO SPECIFIC SUGARS
ABSORPTION
NEONATE, 4608*
PREMATURITY, 4608*
SMALL INTESTINE, 3829
DIARRHEA
DIETARY FACTORS, 5088
DIGESTION
GLUCOSE, 3814
METABOLISM
CIRCULATION, 3811*
FATTY ACIDS, 3930*
LIVER, 3811*, 4007*
MICROSOMES, 4007*
NEONATE, 4608*
PREMATURITY, 4608*

CARBON DIOXIDE
NUCLEIC ACIDS
LIVER, 4022
METABOLISM, 4022

CARBON TETRACHLORIDE
BILE DUCT
KINETICS, CELL, 4060
LIVER
MORPHOLOGY, 4073
PATHOLOGY, 4011*
PHENOBARBITAL, 4011*
REGENERATION, 4060
LIVER INJURY
BILIRUBIN, 4124
ISOENZYMES, 4061
LYSOSOMES, 4110
PREVENTION, 4059

CARCINOGENS
LIVER
ION TRANSPORT, 4148
MITOSIS, 4034
PATHOLOGY, 4011*
NUCLEIC ACIDS
SYNTHESIS, 3993, 4081

CARCINOGENS (continued)
PANCREAS
PATHOLOGY, 3993
PROTEIN
SYNTHESIS, 4081

CARCINOIDS
ENDOCRINE SYSTEM
GASTROINTESTINAL TRACT, 4284
RADIOLOGY, 4284
ESOPHAGUS
NEOPLASMS, 4350
GASTROINTESTINAL
DIAGNOSIS, 5120
ETIOLOGY, 5120
STOMACH
PATHOLOGY, 4472

CATALASE
ALCOHOL
OXIDATION, 3997*
DEFICIENCY
LIVER, 4114
LIVER, 4002*
FATTY LIVER, 4787*
NEOPLASMS, MALIGNANT, 4787*

CATECHOLAMINES
GLYCOGEN
SALIVARY GLANDS, 3912
SMALL INTESTINE
MOTILITY, 3874*
STOMACH
MOTILITY, 3871*, 3874*
URINE
PEPTIC ULCER, 4536

CATHARTICS
SEE LAXATIVES

CECUM
SEE ALSO LARGE INTESTINE
FASCIOLIASIS
ULTRASTRUCTURE, 5158
MOTILITY
ADENOSINE TRIPHOSPHATE, 3907
NEOPLASMS, BENIGN, 4707
ULCER
CYTOMEGALOVIRUS, 4684

CELIAC-AXIS COMPRESSION SYNDROME
ANOMALY, CONGENITAL, 5070
FAMILIAL FACTORS, 5070

CELIAC DISEASE
ANEMIA
REVIEWS, 4634
BLOOD
CHOLECYSTOKININ, 4609*
CHILDREN
DISEASES ASSOCIATED WITH, 4632
EPIDEMIOLOGY, 4627
IMMUNOLOGY, 4632
RADIOLOGY, 4632
REVIEWS, 4632, 4642
COMPLICATIONS
IMMUNOGLOBULINS, 4643
NEOPLASMS, MALIGNANT, 4636, 4643

CELIAC DISEASE (continued)
GALLBLADDER
MOTILITY, 4609*
GLUCOSE
ABSORPTION, 4605*
GLUTEN
INTOLERANCE, 4602*
ILEUM
ABSORPTION, 4605*
IMMUNOGLOBULINS
REVIEWS, 4640
IMMUNOLOGY
REVIEWS, 4640
JEJUNUM
KINETICS, CELL, 4635
PATHOLOGY, 4635
ULTRASTRUCTURE, 4633
LACTOSE INTOLERANCE
DISEASES ASSOCIATED WITH, 4603*
PATHOLOGY
GLUTEN, 4631
PROTEIN
GLUTEN, 4629
IMMUNITY, 4607*
RADIOLOGY
BARIUM, 4630
SMALL INTESTINE
ABSORPTION, 4637
NEOPLASMS, MALIGNANT, 4636
WATER
ABSORPTION, 4605*

CERULEIN
SMALL INTESTINE
MOTILITY, 3863*

CHELATING AGENTS
BILIARY
SURGERY, 4995

CHEMICAL COMPOSITION
ANTRUM
GASTRIN, 3942*
BILE
PORTACAVAL SHUNT, 4136
BLOOD
GASTRIN, 3942*
DUODENUM
GASTRIN, 3942*
GASTRIN
BLOOD, 3973
LIVER
GLYCOGEN, 4079
LIPIDS, 4048
PANCREAS
ENZYMES, 3974*

CHEMORECEPTION
ESOPHAGUS
NERVOUS SYSTEM, 3919

CHEMOTHERAPY
ANUS
NEOPLASMS, MALIGNANT, 4692
BILE DUCT
NEOPLASMS, MALIGNANT, 4947*
COLON
NEOPLASMS, MALIGNANT, 4657*

CHEMOTHERAPY (continued)
ESOPHAGUS
NEOPLASMS, MALIGNANT, 4348
LIVER
NEOPLASMS, MALIGNANT, 4781*, 4788*, 4829

CHENODEOXYCHOLIC ACID
CHOLELITHIASIS
DRUG TREATMENT, 5004

CHILDREN
SEE ALSO NEONATE, AGE FACTORS
CELIAC DISEASE
DISEASES ASSOCIATED WITH, 4632
EPIDEMIOLOGY, 4627
IMMUNOLOGY, 4632
RADIOLOGY, 4632
REVIEWS, 4632, 4642
CROHNS DISEASE
SURGERY, 4720
DIARRHEA, 5031
DRUG TREATMENT, 5020
INFECTION, 5020
REVIEWS, 5020
DYSENTERY
IMMUNOLOGY, 5073
GASTROENTERITIS
ANTIBIOTICS, 5082
GASTROINTESTINAL
ENDOSCOPY, 4291
GASTROINTESTINAL DISEASE
ENDOSCOPY, 4201
GLUTEN
INTOLERANCE, 4602*
HEPATITIS, CHRONIC
IMMUNOSUPPRESSION, 4905*
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4876*
CORTICOSTEROIDS, 4870*
DRUG TREATMENT, 4890
HERNIA
SURGERY, 4584
HERNIA, HIATUS
RADIOLOGY, 4324
INFLAMMATORY BOWEL DISEASE
SURGERY, 4720
LIVER
WILSONS DISEASE, 4785*
LIVER DISEASE
SURGERY, 4834
MALNUTRITION
THERAPY, 5047
MEGACOLON
SURGERY, 4653*
PANCREAS DISEASE
DIAGNOSIS, 4245
PORTAL HYPERTENSION
SURGERY, 4939
SALIVARY GLANDS
NEOPLASMS, 5109, 5111
SALMONELLOSIS
CARRIER STATE, 5072
SMALL INTESTINE
LYMPHANGIECTASIA, 4544*
STOMACH
ACID SECRETION, 4215
ULCERATIVE COLITIS
SURGERY, 4720

CHLORIDE
TRANSPORT
BICARBONATE SECRETION, 4152*
GALLBLADDER, 3856

CHOLANGIOGRAPHY
SEE ANGIOGRAPHY

CHOLANGITIS
PARASITES AND PARASITIC DISEASE
SURGERY, 5011
PATHOLOGY, 4953*
SURGERY
SEQUELAE, 5011

CHOLECYSTECTOMY
ABDOMEN
DRAINAGE, 4972
BILE ACIDS
EXCRETION, 4133
METABOLISM, 4133
CHOLECYSTITIS, 4982
CHOLELITHIASIS
COMPLICATIONS, 4991
GALLBLADDER DISEASE
COMPLICATIONS, 4978
GASTROINTESTINAL
MOTILITY, 3879
SEQUELAE, 4949*
BILE DUCT, 4956

CHOLECYSTITIS
BILE DUCT
RADIOLOGY, 4940*
BILIARY
DRAINAGE, 4984
CHOLECYSTECTOMY, 4982
COMPLICATIONS
AGE FACTORS, 4976
JAUNDICE, OBSTRUCTIVE, 4976
DIAGNOSIS, 4974
EMPHYSEMA
MICROORGANISMS, 4943*
ENDOSCOPY, 4296
GALLBLADDER
CIRCULATION, 4135
NERVOUS SYSTEM, 4135
INTEGUMENTARY SYSTEM
TEMPERATURE, 4537
PARASITES AND PARASITIC DISEASE
SURGERY, 5011
RADIOLOGY
SURGERY, 4988
SIMULATION
DRUG TREATMENT, 4134
VITAMIN C, 4134
SURGERY
AGE FACTORS, 4981
SEQUELAE, 5011

CHOLECYSTOKININ
BLOOD
CELIAC DISEASE, 4609*
CHOLELITHIASIS
DIAGNOSIS, 4945*
ESOPHAGUS
MOTILITY, 3905

CHOLECYSTOKININ (continued)
GALLBLADDER DISEASE
RADIOLOGY, 4286
PANCREAS
SECRETION, 3986

CHOLEDOCHOLITHIASIS
BILE DUCT
STRICTURE, 4988
BLOOD
CLOTTING, 4980
SURGERY
RECURRENCE, 4959
TECHNIQUES, 4946*

CHOLELITHIASIS
AGE FACTORS
COMPLICATIONS, 4969
SURGERY, 4969
ANGIOGRAPHY, 5000
BILE
BILE ACIDS, 4950*
BILE ACIDS
METABOLISM, 4125, 5002, 5009
CHOLECYSTECTOMY
COMPLICATIONS, 4991
CHOLESTEROL, 5003
COMPLICATIONS, 5010
FISTULA, 4954*
CROHNS DISEASE
DISEASES ASSOCIATED WITH, 5193
DIAGNOSIS, 4999
CHOLECYSTOKININ, 4945*
DRUG TREATMENT
CHENODEOXYCHOLIC ACID, 5004
EPIDEMIOLOGY
AGE FACTORS, 4960
DIETARY FACTORS, 4960
GENETIC FACTORS, 4960
SEX FACTORS, 4960
ETIOLOGY, 5000, 5003
FATTY LIVER, 5000
FISTULA
SURGERY, 4954*
GALLBLADDER
MOTILITY, 4944*
LIPIDS
BLOOD, 5001
ORAL CONTRACEPTIVES
EPIDEMIOLOGY, 4951*
PANCREATITIS
DISEASES ASSOCIATED WITH, 4744*
PHOSPHOLIPIDS, 5003
SURGERY, 5010
COMPLICATIONS, 4989
SEQUELAE, 4962, 4989
TECHNIQUES, 4962
THYROID
COMPLICATIONS, 4997
WHIPPLES DISEASE
DISEASES ASSOCIATED WITH, 4625

CHOLERA
ETIOLOGY
REVIEWS, 5037
PREVENTION
DRUGS, 5038

CHOLERA (continued)
SMALL INTESTINE
GLYCOPROTEIN, 5074
IMMUNOGLOBULINS, 5074
PATHOLOGY, 5102
THERAPY, 5040
REVIEWS, 5037

CHOLERETIC AGENTS
BILE
SECRETION, 4806
SYNTHESIS, 4070

CHOLESTASIS
ANTIDEPRESSANTS
METABOLISM, 4138
ANTITRYPSIN, ALPHA
DEFICIENCY, 4838
BILE
EXCRETION, 4138
DRUG METABOLISM
LIVER, 4138
DRUG TREATMENT
PHENOBARBITAL, 4942*
HEPATITIS, NONVIRAL
DRUG-INDUCED, 4862*
LIVER DISEASE, ALCOHOLIC
DISEASES ASSOCIATED WITH, 4916
PREGNANCY
LIVER FUNCTION TESTS, 4955*
RECURRENCE
FAMILIAL FACTORS, 4843
STIMULATION
BILE ACIDS, 4000*

CHOLESTEROL
BILE ACIDS
METABOLISM, 4125
SYNTHESIS, 4129
BLOOD
DRUG EFFECTS ON, 5108
CHOLELITHIASIS, 5003
GALLSTONE
ETIOLOGY, 4941*
SECRETION
BILE ACIDS, 4009*
STOMACH
PSEUDOTUMOR, 4455
SYNTHESIS
LIVER, 4043
PROTEIN, 4043
TRANSPORT
LIVER, 4045

CHOLINERGIC AGENTS
ESOPHAGUS
SPHINCTER, 4486*
STOMACH
ACID SECRETION, 3956
SECRETION, 3926*

CHOLINESTERASE
SMALL INTESTINE
ABSORPTION, 4158
STOMACH
VAGOTOMY, 3971

CHYMOTRYPSIN
FECES
PANCREAS DISEASE, 4246, 4247
PANCREATITIS, 4246

CIRCADIAN RHYTHM
BLOOD
GASTRIN, 3932*
INTESTINE
MITOSIS, 4179
SMALL INTESTINE
ALKALOIDS, 4156
KINETICS, CELL, 4155*

CIRCULATION
AMINO ACIDS
METABOLISM, 3811*, 4852
CARBOHYDRATE
METABOLISM, 3811*
CIRRHOSIS
DRUG TREATMENT, 4936
COMPLICATIONS
LIVER DISEASE, 4852
DUODENUM
PANCREATITIS, 4165*
ELECTROLYTE
LIVER DISEASE, 4103
GALLBLADDER
CHOLECYSTITIS, 4135
GASTROINTESTINAL
ENDOSCOPY, 4211
HORMONE, 3872*
KIDNEY
LIVER DISEASE, 4103
LIVER, 4032
CIRRHOSIS, 4837, 4922*
JAUNDICE, OBSTRUCTIVE, 4987
MARKER STUDIES, 4816
MORPHOLOGY, 4033
PORTACAVAL SHUNT, 4040, 4933
LYMPH
PORTAL HYPERTENSION, 4261
PANCREAS
ELECTRICAL CONTROL, 4166*
PANCREATITIS, CHRONIC, 4745*
PANCREATITIS, CHRONIC
RECURRENCE, 4763
PENTAGASTRIN
COMPLICATIONS, 3955
PROTEIN
METABOLISM, 4852
SMALL INTESTINE
SHOCK, 4168*
STOMACH
DIETARY FACTORS, 3931*
PERMEABILITY, 3844
ULCER, 3940*
ULCERATIVE COLITIS
PATHOLOGY, 3805
RADIOLOGY, 3805

CIRRHOSIS
ABSORPTION
XYLOSE, 4930
ALCOHOLISM
AUTOIMMUNITY, 4917
ANEMIA, 4926

CIRRHOSIS (continued)
ANTIGEN, AUSTRALIA
COMPLEMENT, 4902*
EPIDEMIOLOGY, 4814
ANTITRYPSIN, ALPHA
DEFICIENCY, 4934
ASCITES
ETIOLOGY, 4928
PHOSPHOLIPASE, 4741*
SURGERY, 4932
SURVIVAL, 4937
BLOOD
PHOSPHOLIPID, 4923*
SODIUM, 4919*
VITAMIN A, 4262
CIRCULATION
DRUG TREATMENT, 4936
COMPLICATIONS
BLEEDING, 4927
GASTROENTERITIS, 4927
SURGERY, 4938
DIAGNOSIS, 4266
GALACTOSE, 4198*
IMMUNOGLOBULINS, 4914
TRANSFERRIN, 4914
DIGESTION
XYLOSE, 4930
DISEASES ASSOCIATED WITH
RESPIRATORY SYSTEM, 4934
DIURETICS, 4919*
DUODENUM
PATHOLOGY, 5099
ESOPHAGUS
BLEEDING, 4932, 4938
FETOPROTEIN, ALPHA
EPIDEMIOLOGY, 4814
GASTROINTESTINAL
BLEEDING, 4264
HEPATITIS, VIRAL
ANGIOGRAPHY, 4881
IMMUNOGLOBULINS, 4801, 4802, 4921*
IMMUNOLOGY, 5184
ETIOLOGY, 4925*
LIVER
CIRCULATION, 4837, 4922*
LIVER COMA
INFECTION, BACTERIA, 4912
NERVOUS SYSTEM
DRUG TREATMENT, 4936
PORTACAVAL SHUNT
IMMUNITY, 4924*
IMMUNOLOGY, 4924*
SMALL INTESTINE
DISACCHARIDASE, 5090
ELECTROLYTE, 5053
STOMACH
IMMUNOGLOBULINS, 4429
NEOPLASMS, MALIGNANT, 4927
PATHOLOGY, 5099
ULCER
BLEEDING, 4927
URINE
OXALATE, 5041
VITAMIN A
METABOLISM, 4618

CIRRHOSIS, BILIARY
ANTITRYPSIN, ALPHA
DEFICIENCY, 4838
AUTOIMMUNITY
HEPATITIS, CHRONIC, 4796*

CLOTTING
BLOOD
BILIARY DISEASE, 4980
CHOLEDOCHOLITHIASIS, 4980
HEPATITIS, CHRONIC, 4899*
HEPATITIS, VIRAL, 4885, 4886, 4887
LIVER DISEASE, 4980
PANCREATITIS, 4753
VITAMIN K
LIVER, 4008*

COENZYMES
AMINO ACIDS
METABOLISM, 4091
BILE
LIPIDS, 3975*
LIVER
PHOSPHORYLATION, OXIDATIVE, 4095
ULTRASTRUCTURE, 4113
PANTOTHENIC ACID
METABOLISM, 4075
VITAMIN A
DEFICIENCY, 4113
VITAMIN E
DEFICIENCY, 4113

COLITIS
SEE ALSO ULCERATIVE COLITIS
DRUG INDUCED
ANTIBIOTICS, 4655*, 4701, 4702
INFLAMMATORY BOWEL DISEASE
AMEBIASIS, 5131*
ISCHEMIA
COMPLICATIONS, 4703
GALLBLADDER DISEASE, 4703
PERFORATION, 4703
PRECANCER
CROHNS DISEASE, 5196
RECTUM
BIOPSY, 4655*

COLITIS, GRANULOMATOUS
THERAPY, 4704

COLON
SEE ALSO LARGE INTESTINE
ABSORPTION
REVIEWS, 3840
AMMONIA
TRANSPORT, 4654*
BACTERIA, 4169
BLEEDING
CROHNS DISEASE, 5179
CONTRAST MEDIA
RADIOLOGY, 4240
CROHNS DISEASE, 5186
ANEMIA, 5179
RECURRENCE, 5189
DILATATION
CROHNS DISEASE, 5176, 5177
DISEASES
EPIDEMIOLOGY, 4658

COLON (continued)
DIVERTICULITIS, 4670
DIVERTICULUM, 4670, 4671
ELECTROLYTE
TRANSPORT, 3841
ENDOSCOPY
BIOPSY, 4239
BLEEDING, 4666
COMPLICATIONS, 4666
CROHNS DISEASE, 5185
PERFORATION, 4666
TECHNIQUES, 4236
ENZYMES
VAGOTOMY, 3915
FIBER
DEFICIENCY, 4658
FISTULA
SIMULATION, 4675
INTUSSUSCEPTION, 4691
MOTILITY
BIOPOTENTIALS, 3904
BRADYKININ, 3864*
DRUG EFFECTS ON, 3903
ELECTROPHYSIOLOGY, 3910
NERVOUS CONTROL, 3864*
PROSTAGLANDIN, 3899, 3903
REVIEWS, 3908
NEOPLASMS
IMMUNITY, 4690
SURGERY, 4690
NEOPLASMS, BENIGN
ENDOSCOPY, 4238
NEOPLASMS, MALIGNANT
ANTIGEN, CARCINOEMBRYONIC, 4244, 4646*
CHEMOTHERAPY, 4657*
COMPLICATIONS, 4695
CROHNS DISEASE, 5193
DIAGNOSIS, 4699
DRUG TREATMENT, 4657*
ENDOSCOPY, 4237, 4238
EPIDEMIOLOGY, 4688, 4699
ETIOLOGY, 4699
IMMUNOLOGY, 4648*, 4687
INFECTION, 4694
ISCHEMIA, 4695
KIDNEY, 4698
PROGNOSIS, 4648*
RECURRENCE, 4687, 4689
SURGERY, 4649*
POLYPS
ANGIOGRAPHY, 4677
ENDOSCOPY, 4676
FAMILIAL FACTORS, 4678
LYMPHATICS, 4680
PRESSURE STUDIES
CONSTIPATION, 4651*
DIARRHEA, 4651*
RADIOLOGY
TECHNIQUES, 4243
SECRETION
REVIEWS, 3840
SMALL INTESTINE
CROHNS DISEASE, 5193
STENOSIS
DISEASES ASSOCIATED WITH, 4664
PANCREATITIS, 4664

COLON (continued)
ULCER
DIAGNOSIS, 4659
UREA
TRANSPORT, 4654*
WATER
TRANSPORT, 3841

COMPLEMENT
CIRRHOSIS
ANTIGEN, AUSTRALIA, 4902*

COMPUTERS
DYSPEPSIA
DIAGNOSIS, 4297
GASTROINTESTINAL
ENDOSCOPY, 4205

CONSTIPATION
COLON
PRESSURE STUDIES, 4651*

CONTRAST MEDIA
BILIARY
ANGIOGRAPHY, 4279
COLON
RADIOLOGY, 4240
ESOPHAGUS
PERFORATION, 4195*
RUPTURE, 4195*
LIVER
ANGIOGRAPHY, 4106
STOMACH
RADIOLOGY, 4220

COPPER
ACRODERMATITIS ENTEROPATHICA, 5128
KINETICS
WILSONS DISEASE, 4782*
METABOLISM
LIVER, 4082
RADIOPROTECTIVE AGENTS, 4082

CORTICOSTEROIDS
GASTRITIS
DRUG-INDUCED, 3920*
HEPATITIS, CHRONIC
DRUG TREATMENT, 4910
LYMPHOCYTE, 4871*
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4870*, 4882
CHILDREN, 4870*
LYMPHOCYTE, 4871*
LIVER
AGE FACTORS, 4096
GLUCONEOGENESIS, 4077
ULTRASTRUCTURE, 4065
METABOLISM
LIVER, 4097
NUCLEIC ACIDS
BILIARY TRACT, 4131
SYNTHESIS, 4131
PROTEIN
SYNTHESIS, 4065
UREA
SYNTHESIS, 4094

CRIGLER-NAJJAR SYNDROME
PHENOBARBITAL, 4856
PHOTOTHERAPY, 4856

CROHNS DISEASE
ANEMIA, 5172*
ANORECTUM
STRICTURE, 5163*
ANUS
ABSCESS, 5192
FISSURE, 5194
FISTULAS, 5192
ULCER, 5192
BIOPSY
RECTUM, 5181
BLEEDING
RECURRENCE, 5187
BLOOD
VITAMIN A, 4262
CHILDREN
SURGERY, 4720
COLITIS
PRECANCER, 5196
COLON, 5186
ANEMIA, 5179
BLEEDING, 5179
DILATATION, 5176, 5177
ENDOSCOPY, 5185
NEOPLASMS, MALIGNANT, 5193
RECURRENCE, 5189
COMPLICATIONS, 5171*
BLIND LOOP SYNDROME, 5195
DILATATION, 5177
RECTUM, 5179
DIAGNOSIS
SURGERY, 5194
DIARRHEA, 5172*
DISEASES ASSOCIATED WITH
CHOLELITHIASIS, 5193
MUSCULOSKELETAL SYSTEM, 5170*
PANCREATITIS, 5193
DRUG TREATMENT
ANEMIA, 5195
FOLIC ACID, 5195
IMMUNOSUPPRESSION, 5188
IRON, 5195
VITAMIN B12, 5195
ENDOSCOPY, 5174*, 5189
EPIDEMIOLOGY, 5178
ESOPHAGUS
ENDOSCOPY, 5185
GASTROINTESTINAL
BLEEDING, 5187
ILEUM
RECURRENCE, 5189
IMMUNOGLOBULINS
LARGE INTESTINE, 5166*
SMALL INTESTINE, 5166*
IMMUNOLOGY, 5170*
INTESTINE
PATHOLOGY, 5190
JEJUNUM
BIOPSY, 5162*
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5180
MALABSORPTION
VITAMIN D, 5195
RADIOLOGY, 5172*
RECURRENCES, 5171*
REVIEWS, 4712, 5183
SMALL INTESTINE
COLON, 5193
ENDOSCOPY, 5185

CROHNS DISEASE (continued)
SMALL INTESTINE (continued)
NEOPLASMS, MALIGNANT, 5180
OBSTRUCTION, 5193
SODIUM
TRANSPORT, 5164*
STOMACH
ENDOSCOPY, 5185
RADIOLOGY, 5168*
SURGERY, 5190
RECURRENCE, 5174*, 5189
SEQUELAE, 4719
TECHNIQUES, 5182
THYROID
HORMONE, 5160*, 5161*
IODINE, 5165*
ULCERATIVE COLITIS
DIAGNOSIS, 5194
VITAMIN A
METABOLISM, 4618

CRYOSURGERY
SMALL INTESTINE
HEALING, 4408
STOMACH
HEALING, 4408

CYSTIC FIBROSIS
LIVER FUNCTION TESTS, 4920*
PANCREAS FUNCTION TESTS, 4734

CYSTS
BILE DUCT, 4956
DRAINAGE, 4985
PERMEABILITY, 4973
ECHINOCOCCOSIS
COMPLICATIONS, 5139
GALLBLADDER
RUPTURE, 4979
TRAUMA, 4979
LIVER
ANGIOGRAPHY, 4869
DIAGNOSIS, 4824
PANCREAS
COMPLICATIONS, 4729
ETIOLOGY, 4770
RADIOLOGY, 4743*
SURGERY, 4770

CYTOCHROMES
DRUG METABOLISM
LIVER, 4051
LIVER
AGE FACTORS, 4083
SEX FACTORS, 4083

CYTOLOGY
LARGE INTESTINE
DISEASES, 4241
LIVER, 4298
STOMACH
NEOPLASMS, MALIGNANT, 4224, 4389*

CYTOMEGALOVIRUS
CECUM
ULCER, 4684
LARGE INTESTINE
PATHOLOGY, 5071

DEATH
SEE SURVIVAL

DEFECATION
PRESSURE STUDIES
MEGACOLON, 4645*

DEGLUTITION
DYSKINESIA, 4310
MERYCISM
REVIEWS, 4309
NERVOUS CONTROL, 4310
SPASM, 4310

DEHYDRATION
LIVER
ULTRASTRUCTURE, 4053

DEHYDROGENASE
LIVER
VITAMIN B1, 4086

DERMATITIS HERPETIFORMIS
REVIEWS, 4639

DETOXIFICATION
ENDOTOXIN
LIVER, 4066

DIABETES
LIVER
FATTY ACIDS, 4844
TRIGLYCERIDES, 4844

DIAPHRAGM
HERNIA
DIAGNOSIS, 4326
SURGERY, 4326

DIARRHEA
CHILDREN, 5031
DRUG TREATMENT, 5020
INFECTION, 5020
REVIEWS, 5020
COLON
PRESSURE STUDIES, 4651*
CROHNS DISEASE, 5172*
DIETARY FACTORS
CARBOHYDRATE, 5088
DISACCHARIDASE
INTOLERANCE, 5045
DRUG-INDUCED
ANTIBIOTICS, 5013*
LAXATIVES, 5083
TRANQUILIZERS, 5079
ETIOLOGY
SULFATE, 5097
FIBRINOGEN
DEFICIENCY, 5034
GALLBLADDER
NEOPLASMS, MALIGNANT, 4994
NEONATE, 5031
LACTATE DEHYDROGENASE, 5050
NUTRITION, 5018*
ZOLLINGER-ELLISON SYNDROME
HYPOCHLORHYDRIA, 5121

DIET
KWASHIORKOR
THERAPY, 5016*

DIETARY FACTORS
ABSORPTION, 3806*
APPENDICITIS
FIBER, 5089
BILE ACIDS
SYNTHESIS, 4129
BLOOD
GASTRIN, 3931*
CALCIUM
ABSORPTION, 3845
CHOLELITHIASIS
EPIDEMIOLOGY, 4960
DIARRHEA
CARBOHYDRATE, 5088
DIVERTICULUM
FIBER, 5089
FECES
PROTEIN, 5092
GASTRITIS
ETIOLOGY, 4375*
GLUCOSE
ABSORPTION, 4153*
DIGESTION, 4153*
HEMORRHOIDS
FIBER, 5089
HERNIA, HIATUS
FIBER, 5089
ILEOSTOMY
DRAINAGE, 4709*
LIPIDS
SYNTHESIS, 4150*
LIVER
LIPIDS, 4047
NECROSIS, 4071
MALABSORPTION
DRUG-INDUCED, 4641
PROTEIN
SYNTHESIS, 5096
REGIONAL ENTERITIS, 5167*
SMALL INTESTINE
GLYCOLYSIS, 4161
STOMACH
ACID SECRETION, 3931*
CIRCULATION, 3931*
ULCER
STRESS, 3921*

DIGESTION
CARBOHYDRATE
GLUCOSE, 3814
CIRRHOSIS
XYLOSE, 4930
GASTRECTOMY
SEQUELAE, 4410
GASTROINTESTINAL
MORPHOLOGY, 3820
GLUCOSE
DIETARY FACTORS, 4153*
NITROGEN
LACTOSE, 4153*
PROTEIN
LACTOSE, 4153*
SMALL INTESTINE
ENTEROCOLITIS, 4597
STOMACH, 4418

DISACCHARIDASE
SEE ALSO LACTASE
DEFICIENCY
INFECTION, 5098
SMALL INTESTINE, 5098
INTOLERANCE
DIARRHEA, 5045
NEONATE, 5045
PROTEIN
DEFICIENCY, 5090
SMALL INTESTINE
ALCOHOL, 4151*
ANTINEOPLASTIC AGENTS, 4157
CIRRHOSIS, 5090
MALABSORPTION, 5049
PROTEIN, 5090
STOMACH
PRECANCER, 4387*

DISACCHARIDE
MALABSORPTION
ANTINEOPLASTIC AGENTS, 4157
SMALL INTESTINE
ALCOHOLS, POLYHYDRIC, 4162

DIURETICS
ASCITES, 4919*
BLOOD
SODIUM, 4919*
CIRRHOSIS, 4919*
ION TRANSPORT
SMALL INTESTINE, 4152*

DIVERTICULITIS
COLON, 4670

DIVERTICULUM
COLON, 4670, 4671
DIETARY FACTORS
FIBER, 5089
ESOPHAGUS, 4332, 4333
DYSKINESIA, 4331
MECKELS DIVERTICULUM
MORPHOLOGY, 4599

DRAINAGE
ABDOMEN
CHOLECYSTECTOMY, 4972
BILE DUCT
CYSTS, 4985
NEOPLASMS, 4977
BILIARY
CHOLECYSTITIS, 4984
COMPLICATIONS, 4983
TECHNIQUES, 4983, 4984
GALLBLADDER DISEASE
COMPLICATIONS, 4978
ILEOSTOMY
DIETARY FACTORS, 4709*
PANCREAS
PSEUDOCYST, 4776
PSEUDOCYST
PANCREAS, 4728

DRUG ABSORPTION
GASTROINTESTINAL TRACT, 3821
INTESTINE, 3822
SMALL INTESTINE
ALCOHOL, 3858
ANTINEOPLASTIC AGENTS, 3860
STARVATION, 3860
VITAMIN B2, 3824

DRUG-INDUCED
COLITIS
ANTIBIOTICS, 4655*, 4701, 4702
DIARRHEA
ANTIBIOTICS, 5013*
LAXATIVES, 5083
TRANQUILIZERS, 5079
GASTRITIS
CORTICOSTEROIDS, 3920*
GASTROINTESTINAL
BLEEDING, 4374*, 4421, 4437
HEPATITIS, NONVIRAL
ANESTHETICS, 4863
ANTIBACTERIALS, 4858*, 4859*, 4864
CHOLESTASIS, 4862*
RISK FACTORS, 4859*
TRANSFUSION, 4864
LARGE INTESTINE
OBSTRUCTION, 4669
LIVER COMA
ANTIBACTERIALS, 4865
ANTIBIOTICS, 4865
LIVER INJURY
ANESTHETICS, 4857*
ANTIBACTERIALS, 4867
MALABSORPTION
ALCOHOL, 4641
DIETARY FACTORS, 4641
PATHOLOGY, 4641
REVIEWS, 4641
PANCREATITIS
ANTINEOPLASTIC AGENTS, 4758
PEPTIC ULCER
AGE FACTORS, 4538
ANTIINFLAMMATORY AGENTS, 4500*, 4538
HEALING, 4500*
VITAMIN C, 4500*
STOMACH
BLEEDING, 4374*, 4424
ULCER, 3929*

DRUG METABOLISM
CYTOCHROMES
LIVER, 4051
ENZYMES
DRUG EFFECTS ON, 4059
KINETICS
LIVER, 4056
LIVER
ALCOHOL, 4010*
ANTIBIOTICS, 4010*
CADMIUM, 4058
CHOLESTASIS, 4138
EMBRYOLOGY, 3787*
HYPOLIPEMIC AGENTS, 3854
ULTRASTRUCTURE, 3787*
ZINC, 4057

DRUG METABOLISM (continued)
MICROSOMES
LIVER, 4052
TRANQUILIZERS
LIVER DISEASE, 4793*

DRUG TOXICITY
INFLAMMATORY BOWEL DISEASE
DRUG TREATMENT, 4182
PEPTIC ULCER
DRUG TREATMENT, 4182
SMALL INTESTINE
DRUG TREATMENT, 4182

DRUG TREATMENT
ACRODERMATITIS ENTEROPATHICA
ZINC, 5113
AMEBIASIS, 5147
ASCARIASIS, 5143
ASCITES, 4931
CHOLECYSTITIS
STIMULATION, 4134
CHOLELITHIASIS
CHENODEOXYCHOLIC ACID, 5004
CHOLESTASIS
PHENOBARBITAL, 4942*
CIRRHOSIS
CIRCULATION, 4936
NERVOUS SYSTEM, 4936
COLON
NEOPLASMS, MALIGNANT, 4657*
CROHNS DISEASE
ANEMIA, 5195
FOLIC ACID, 5195
IMMUNOSUPPRESSION, 5188
IRON, 5195
VITAMIN B12, 5195
DIARRHEA
CHILDREN, 5020
DUODENUM
STENOSIS, 4550
ESOPHAGITIS, REFLUX, 4356
ESOPHAGUS DISEASE
ANTACIDS, 4302
GASTRECTOMY
SEQUELAE, 4404
GASTRITIS, 4427
GASTROINTESTINAL
BLEEDING, 5060
HEPATITIS
STIMULATION, 4108
HEPATITIS, CHRONIC
COMPLICATIONS, 4907
CORTICOSTEROIDS, 4910
IMMUNOSUPPRESSION, 4906*
HEPATITIS, VIRAL
CHILDREN, 4890
INFLAMMATORY BOWEL DISEASE
DRUG TOXICITY, 4182
LARGE INTESTINE
OBSTRUCTION, 4668
LIVER
AMEBIASIS, 5149
LIVER COMA, 4935
LIVER DISEASE, 4800
COMPLICATIONS, 4805
MALNUTRITION
COMPLICATIONS, 5023

DRUG TREATMENT (continued)
PANCREATITIS
ANTIBIOTICS, 4739*
ANTIHISTAMINES, 4756
BLEEDING, 4750
PARASITES AND PARASITIC DISEASE, 5143
PEPTIC ULCER, 4518
CARBENOXALONE, 4370*
DRUG TOXICITY, 4182
PREVENTION, 4496*
SIMULATION, 4501*
SALMONELLOSIS, 5058
SCHISTOSOMIASIS, 4935, 5150
COMPLICATIONS, 5132*
SMALL INTESTINE
DRUG TOXICITY, 4182
STOMACH
FOREIGN BODIES, 4417
STOMACH DISEASE
ANTACIDS, 4302
DYSPEPSIA, 4392

DUMPING SYNDROME
GASTRECTOMY
TECHNIQUES, 4401

DUODENITIS
DIAGNOSIS
TECHNIQUES, 4590
EROSIONS
BLEEDING, 4426
PHENYLKETONURIA
DISEASES ASSOCIATED WITH, 5103

DUODENUM
SEE ALSO SMALL INTESTINE
ABSORPTION
FATTY ACIDS, 3791*
ULTRASTRUCTURE, 3791*
BIOPOTENTIALS
VAGOTOMY, 3883
BIOPSY
GASTROINTESTINAL DISEASE, 5051
PANCREATITIS, 5051
ULCER, 5051
BLEEDING
SURGERY, 5085
CIRCULATION
PANCREATITIS, 4165*
DISEASES, 4302
RADIOLOGY, 4233
ENDOSCOPY, 4229, 4230, 4591
REVIEWS, 4209
ENTEROKINASE
MALABSORPTION, 4612*
ENZYMES
VAGOTOMY, 3915
GASTRIN
CHEMICAL COMPOSITION, 3942*
HEMATOMA
SURGERY, 4568
HODGKINS DISEASE
BLEEDING, 4557
PERFORATION, 4557
HORMONES
BIOPOTENTIALS, 3831
IRON
ABSORPTION, 3851

DUODENUM (continued)
MOTILITY
BIOPOTENTIALS, 3901
ELECTRICAL CONTROL, 3901
PANCREAS DISEASE, 3882
VAGOTOMY, 3883
NEOPLASMS, BENIGN, 4551
NEOPLASMS, MALIGNANT
ETIOLOGY, 4598
PANCREAS
HETEROTOPIA, 4733
PATHOLOGY
CIRRHOSIS, 5099
HEPATITIS, CHRONIC, 5099
POLYPS
RADIOLOGY, 4552
RADIOLOGY
ANALGESICS, 4411
TECHNIQUES, 4234
REGIONAL ENTERITIS, 5169*
STENOSIS
DRUG TREATMENT, 4550
ETIOLOGY, 4550
SURGERY, 4550
ULCER
CARBENOXALONE, 4503*

DYSENTERY
IMMUNOLOGY
CHILDREN, 5073

DYSKINESIA
DEGLUTITION, 4310
ESOPHAGUS
DIVERTICULUM, 4331
RADIOLOGY, 4317
REVIEWS, 4305
STOMACH
REVIEWS, 4305

DYSPEPSIA
DIAGNOSIS
COMPUTERS, 4297
STOMACH DISEASE
DRUG TREATMENT, 4392

DYSPHAGIA
DIAGNOSIS
PRESSURE STUDIES, 4217
ENDOSCOPY, 4216
PRESSURE STUDIES, 4300*
RADIOLOGY, 4300*
SURGERY, 4319

ECHINOCOCCOSIS
BILE DUCT
OBSTRUCTION, 5139
CYSTS
COMPLICATIONS, 5139
LIVER
COMPLICATIONS, 5139
DIAGNOSIS, 5148
SURGERY, 5148
REVIEWS, 5138

ECHOGRAPHY
LIVER
NEOPLASMS, MALIGNANT, 4822
PSEUDOCYST
PANCREAS, 4727

ELECTRICAL CONTROL
DUODENUM
MOTILITY, 3901
GASTROINTESTINAL
MOTILITY, 3879
ILEUM
MOTILITY, 3870*
PANCREAS
CIRCULATION, 4166*

ELECTROLYTE
ABSORPTION
GASTRIN, 3839
GLUCAGON, 3815*
INTESTINE, 3830
JEJUNUM, 3815*
CIRCULATION
LIVER DISEASE, 4103
LARGE INTESTINE
FISTULA, 4672
SMALL INTESTINE
CIRRHOSIS, 5053
ENTEROCOLITIS, 5053
GASTRITIS, 5053
HEPATITIS, CHRONIC, 5053
PANCREATITIS, CHRONIC, 5053
PEPTIC ULCER, 5053
ULCERATIVE COLITIS, 5053
STOMACH
ATROPINE, 4499*
TRANSPORT
COLON, 3841
DRUG EFFECTS ON, 3833
LAXATIVES, 3841

ELECTROPHORESIS
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4896
LIVER
NUCLEIC ACIDS, 4173
PARASITES AND PARASITIC DISEASE
ANTIGEN, 4193*
DIAGNOSIS, 4193*
STOMACH
NUCLEIC ACIDS, 4173

ELECTROPHYSIOLOGY
COLON
MOTILITY, 3910
INTESTINE
MOTILITY, 3895
STOMACH
BLEEDING, 3925*
ENDOTOXIN, 3925*
EPINEPHRINE, 3925*

EMBRYOLOGY
ESOPHAGUS

NEOPLASMS, MALIGNANT, 4345
INTESTINE
ULTRASTRUCTURE, 3793*
LIVER
DRUG METABOLISM, 3787*

EMESIS
SEE VOMITING

EMPHYSEMA
CHOLECYSTITIS
MICROORGANISMS, 4943*

ENDOCRINE DISEASE
HEPATITIS
SIMULATION, 4109

ENDOCRINE SYSTEM
CARCINOIDS
GASTROINTESTINAL TRACT, 4284
RADIOLOGY, 4284
GASTROINTESTINAL
NEOPLASMS, 5114, 5116
GASTROINTESTINAL TRACT
MORPHOLOGY, 5117
PATHOLOGY, 5117
NEOPLASMS
GASTROINTESTINAL TRACT, 4283
PATHOLOGY, 5118
RADIOIMMUNOASSAY, 4283
RADIOLOGY, 4284

ENDOSCOPY
BILIARY DISEASE, 4256, 4257, 4272
ANGIOGRAPHY, 4288
CHOLECYSTITIS, 4296
COLON
BIOPSY, 4239
BLEEDING, 4666
COMPLICATIONS, 4666
CROHNS DISEASE, 5185
NEOPLASMS, BENIGN, 4238
NEOPLASMS, MALIGNANT, 4237, 4238
PERFORATION, 4666
POLYPS, 4676
TECHNIQUES, 4236
CROHNS DISEASE, 5174*, 5189
DUODENUM, 4229, 4230, 4591
REVIEWS, 4209
DYSPHAGIA, 4216
ESOPHAGITIS
BIOPSY, 4329
ESOPHAGITIS, REFLUX, 4316
ESOPHAGUS
COMPLICATIONS, 4340
CROHNS DISEASE, 5185
REVIEWS, 4209
STENOSIS, 4218
GALLBLADDER DISEASE
ANGIOGRAPHY, 4287
GASTRITIS, 4423
GASTROINTESTINAL
BLEEDING, 4206, 4208, 4264, 5130
CHILDREN, 4291
CIRCULATION, 4211
COMPUTERS, 4205
FISTULA, 5122
NEOPLASMS, MALIGNANT, 4289
RESPIRATORY SYSTEM, 4211
REVIEWS, 4203
TECHNIQUES, 4221, 4291
GASTROINTESTINAL DISEASE
CHILDREN, 4201
HODGKINS DISEASE
LYMPHATICS, 4261
INTESTINE
METAPLASIA, 4192*

ENDOSCOPY (continued)
NEOPLASMS, MALIGNANT
LYMPHATICS, 4261
PANCREAS DISEASE, 4252, 4255, 4256, 4257
COMPLICATIONS, 4258
SURGERY, 4254
PANCREATITIS, 4252, 4255
PANCREATITIS, CHRONIC
SURGERY, 4254
PAPILLA OF VATER, 4274
PEPTIC ULCER, 4227
SMALL INTESTINE
CROHNS DISEASE, 5185
NEOPLASMS, BENIGN, 4293
STOMACH, 4216
ACIDITY, 4397
CROHNS DISEASE, 5185
DRUG EFFECTS ON, 4381*
EROSIONS, 4435
MACROGLOBULINEMIA, 4473
NEOPLASMS, BENIGN, 4293, 4474
NEOPLASMS, MALIGNANT, 4192*, 4196*, 4224, 4383*, 4448, 4449
PEPTIC ULCER, 4415
POLYPS, 4222, 4371*
REVIEWS, 4209

ENDOTOXIN
DETOXIFICATION
LIVER, 4066
IRRADIATION
TRAUMA, 4180
STOMACH
ELECTROPHYSIOLOGY, 3925*

ENEMAS
BARIUM
LAXATIVES, 4243
TECHNIQUES, 4243

ENTERITIS
SEE ALSO REGIONAL ENTERITIS
ANTRECTOMY
SEQUELAE, 4402
PEPTIC ULCER
VAGOTOMY, 4592
VAGOTOMY
SEQUELAE, 4402
VIRUS
IMMUNOGLOBULINS, 5014*

ENTEROCOLITIS
ANOMALY, CONGENITAL
SURGERY, 5173*
HIRSCHSPRUNGS DISEASE, 4681
INFECTION, BACTERIA, 5033
SIMULATION
HYPOXIA, 5017*
STRESS, 5017*
TEMPERATURE, 5017*
SMALL INTESTINE
ABSORPTION, 4597
DIGESTION, 4597
ELECTROLYTE, 5053
STOMACH
HETEROTOPIA, 4450
TRANSMISSION, 5105
YERSINIA, 5119, 5127

ENTEROCOLITIS, NECROTIZING
NEONATE
SURGERY, 5084
THERAPY, 5084
REVIEWS, 5080

ENTEROKINASE
DEFICIENCY
FAMILIAL FACTORS, 4540*
SMALL INTESTINE, 4540*
DUODENUM
MALABSORPTION, 4612*
JEJUNUM
MALABSORPTION, 4612*

EPIDEMIOLOGY
CELIAC DISEASE
CHILDREN, 4627
CHOLELITHIASIS
AGE FACTORS, 4960
DIETARY FACTORS, 4960
GENETIC FACTORS, 4960
ORAL CONTRACEPTIVES, 4951*
SEX FACTORS, 4960
CIRRHOSIS
ANTIGEN, AUSTRALIA, 4814
FETOPROTEIN, ALPHA, 4814
COLON
DISEASES, 4658
NEOPLASMS, MALIGNANT, 4688, 4699
CROHNS DISEASE, 5178
GALLBLADDER
ANOMALY, CONGENITAL, 4971
GASTRITIS, ATROPHIC
ANTRUM, 4481
GASTRITIS, EROSIVE
BLEEDING, 4382*
GASTROENTERITIS
ESCHERICHIA COLI, 5112
HELMINTHIASIS, 5135, 5136, 5141
HEPATITIS, VIRAL
ANTIGEN, 4878
ANTIGEN, AUSTRALIA, 4877, 4882, 4893
INTESTINE
PROTOZOA, 5140
LACTOSE INTOLERANCE, 4644
LIVER
NEOPLASMS, MALIGNANT, 4794*
LIVER DISEASE
FETOPROTEIN, ALPHA, 4815
LIVER INJURY
PREGNANCY, 4868
PANCREATITIS
AGE FACTORS, 4778
RECURRENCE, 4778
SEX FACTORS, 4778
PANCREATITIS, CHRONIC
CALCIFICATION, 4742*
PEPTIC ULCER, 4485*
PROTOZOA
INFECTION, 5135
SALMONELLOSIS, 5030, 5044, 5046
STOMACH
NEOPLASMS, MALIGNANT, 4460, 4467
ULCERATIVE COLITIS, 4713

EPINEPHRINE
LIVER
MITOSIS, 4144
REGENERATION, 4146
STOMACH
ELECTROPHYSIOLOGY, 3925*

EROSIONS
ARTERY
PANCREAS DISEASE, 4729
BILE
REFLUX, 3939*
DUODENITIS
BLEEDING, 4426
SMALL INTESTINE
BLEEDING, 4425
STOMACH, 4434
ASPIRIN, 4388*, 4391*
BLEEDING, 4425
ENDOSCOPY, 4435
SEQUELAE, 4433
STRESS, 3939*

ERYTHROCYTE
BILIRUBIN
BINDING, 4122

ESCHERICHIA COLI
GASTROENTERITIS
EPIDEMIOLOGY, 5112
NEONATE, 5112

ESOPHAGITIS
ENDOSCOPY
BIOPSY, 4329
ESOPHAGUS
PERMEABILITY, 4301*
ETIOLOGY
BILE ACIDS, 4301*

ESOPHAGITIS, REFLUX
ANTACIDS, 4316
COMPLICATIONS
STRICTURE, 4327
SURGERY, 4327
DRUG TREATMENT, 4356
ENDOSCOPY, 4316
ESOPHAGUS
SPHINCTER, 4299*
PRESSURE STUDIES, 4316
RADIOLOGY, 4315
SURGERY
TECHNIQUES, 4328
THERAPY
COMPLICATIONS, 4330

ESOPHAGUS
ACIDITY
PHYSICAL FACTORS, 3862*
ANOMALY
THERAPY, 4307
ATRESIA
SURGERY, 4320
BLEEDING
CIRRHOSIS, 4932, 4938
SURGERY, 4308, 5085
THERAPY, 4184
CHEMORECEPTION
NERVOUS SYSTEM, 3919

ESOPHAGUS (continued)
COMPLICATIONS
GASTROSTOMY, 4365
DISEASES
RADIOLOGY, 4315
DIVERTICULUM, 4332, 4333
DYSKINESIA
DIVERTICULUM, 4331
RADIOLOGY, 4317
REVIEWS, 4305
ENDOSCOPY
COMPLICATIONS, 4340
CROHNS DISEASE, 5185
REVIEWS, 4209
HERNIA
REFLUX, 4314
SIMULATION, 4314
STRANGULATION, 4362
SURGERY, 4314
HERNIA, HIATUS
ULCER, 4313
INFECTION, BACTERIA
DIAGNOSIS, 4311
INFLAMMATION
PSEUDOTUMOR, 4364
MORPHOLOGY
NERVOUS SYSTEM, 3919
MOTILITY
CHOLECYSTOKININ, 3905
NEOPLASMS
CARCINOIDS, 4350
DISEASES ASSOCIATED WITH, 4347
NEOPLASMS, BENIGN
PATHOLOGY, 4344
NEOPLASMS, MALIGNANT
ALCOHOLISM, 5094
CHEMOTHERAPY, 4348
DIAGNOSIS, 4351, 4354
DISEASES ASSOCIATED WITH, 4360, 4447
EMBRYOLOGY, 4345
PROGNOSIS, 4351
RADIOLOGY, 4348
RADIOTHERAPY, 4346, 4348, 4355
RECURRENCE, 4348
SURGERY, 4346, 4349, 4352, 4353
PERFORATION
CONTRAST MEDIA, 4195*
RADIOLOGY, 4340
SURGERY, 4341
PERISTALSIS
NERVOUS CONTROL, 3867*
PERMEABILITY
ACIDS, 4301*
BILE ACIDS, 4301*
ESOPHAGITIS, 4301*
POLYPS
HAMARTOMA, 4343
PRESSURE STUDIES
PHYSICAL FACTORS, 3862*
TECHNIQUES, 3873*
PROLAPSE, 4312
REFLUX
DIAGNOSIS, 4187*
GASTRECTOMY, 4403
HERNIA, HIATUS, 4363
RADIOLOGY, 4363
SURGERY, 4304, 4306

ESOPHAGUS (continued)
RUPTURE
CONTRAST MEDIA, 4195*
ETIOLOGY, 4342
SURGERY, 4341, 4342
SCLERODERMA, 4339
DIAGNOSIS, 4337, 4338
RADIOLOGY, 4338
SPHINCTER
ANTRECTOMY, 4486*
CHOLINERGIC AGENTS, 4486*
ESOPHAGITIS, REFLUX, 4299*
HERNIA, 4318
HORMONE EFFECTS ON, 4318
PRESSURE STUDIES, 3873*, 3905, 4486*
REFLUX, 4318
VAGOTOMY, 4486*
STENOSIS
ABSCESS, 4336
ANOMALY, CONGENITAL, 4358
ENDOSCOPY, 4218
NEONATE, 4336
RADIOLOGY, 4218
REFLUX, 4335
SURGERY, 4334
SURGERY
COMPLICATIONS, 4320, 4357
SEQUELAE, 4306
VARICES
BLEEDING, 4184, 4308, 4938
PSEUDOTUMOR, 4359
RUPTURE, 4938

ESOPHAGUS DISEASE
DRUG TREATMENT
ANTACIDS, 4302
MEDIASTINUM
INFLAMMATION, 4347
SCANNING, SCINTILLATION
RADIOISOTOPES, 4200*
SURGERY, 4303

ETHNIC FACTORS
PEPTIC ULCER
ACID SECRETION, 4504*
STOMACH
SECRETION, 4495*

EXCRETION
ANTIBIOTICS
BILE, 4120
BILE
ANTIDEPRESSANTS, 4138
CHOLESTASIS, 4138
PORTACAVAL SHUNT, 4136
BILE ACIDS
CHOLECYSTECTOMY, 4133
HIRSCHSPRUNGS DISEASE, 4656*
BILIARY
HORMONE, 3852
STEROID, 3857
BILIRUBIN
BILE, 4120
STEROID
SEX FACTORS, 3857
SULFOBROMOPHTHALEIN
BILE, 4120

EXCRETION (continued)
SURFACTANTS
FECES, 3823
URINE, 3823
UROBILINOGEN
REVIEWS, 4116

FAMILIAL FACTORS
ACHALASIA, 4321
CELIAC-AXIS COMPRESSION SYNDROME, 5070
CHOLESTASIS
RECURRENCE, 4843
ENTEROKINASE
DEFICIENCY, 4540*
HEPATITIS, CHRONIC
IMMUNOGLUBULINS, 4903*
POLYPS
COLON, 4678
RECTUM, 4678
SHORT BOWEL SYNDROME
ANOMALY, CONGENITAL, 4547
SMALL INTESTINE
ANOMALY, CONGENITAL, 4547

FASCIOLIASIS
CECUM
ULTRASTRUCTURE, 5158

FATS
SEE LIPIDS, TRIGLYCERIDES

FATTY ACIDS
ABSORPTION, 3824
INTESTINE, 3850
BLOOD
PANCREATITIS, 4755
SECRETIN, 3937*
CARBOHYDRATE
METABOLISM, 3930*
DUODENUM
ABSORPTION, 3791*
LIPIDS
METABOLISM, 3930*
LIVER
DIABETES, 4844
FATTY LIVER, 4844
STARVATION, 4046
OXIDATION
ALCOHOL, 4154*
SMALL INTESTINE, 4154*
SMALL INTESTINE
ABSORPTION, 3849
STOMACH
ACID SECRETION, 3930*
SYNTHESIS
LIVER, 4055

FATTY LIVER
CHOLELITHIASIS, 5000
ETIOLOGY
SHUNT, INTESTINAL, 4001*
LIVER
CATALASE, 4787*
FATTY ACIDS, 4844
TRIGLYCERIDES, 4844
OBESITY
SURGERY, 4795*

FECES
CHYMOTRYPSIN
PANCREAS DISEASE, 4246, 4247
PANCREATITIS, 4246
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4896
PROTEIN
DIETARY FACTORS, 5092
SURFACTANTS
EXCRETION, 3823
VIRUS, 4172

FEEDING
ASBESTOS
PRIMATES, 4176
BLOOD
GASTRIN, 3932*
CALCIUM
ABSORPTION, 4619
LIVER
REGENERATION, 4021
PROTEIN
SALIVARY GLANDS, 3914
STOMACH
ADENYL CYCLASE, 3970
PHOSPHODIESTERASE, 3970
UREA
SYNTHESIS, 4050

FERRITIN
ABSORPTION, 3806*

FETOPROTEIN, ALPHA
CIRRHOSIS
EPIDEMIOLOGY, 4814
LIVER
NEOPLASMS, 4821
NEOPLASMS, MALIGNANT, 4014*, 4814, 4823
LIVER DISEASE
EPIDEMIOLOGY, 4815

FIBER
APPENDICITIS
DIETARY FACTORS, 5089
DEFICIENCY
COLON, 4658
DIVERTICULUM
DIETARY FACTORS, 5089
HEMORRHOIDS
DIETARY FACTORS, 5089
HERNIA, HIATUS
DIETARY FACTORS, 5089

FIBRINOGEN
DEFICIENCY
DIARRHEA, 5034

FIBRINOLYSIS
BLOOD
GASTRITIS, 4394
HEPATITIS, CHRONIC
BLOOD, 4899*
HEPATITIS, VIRAL, 4884, 4885
LIVER, 4008*
NECROSIS, 4886

FIBRINOLYSIS (continued)
STOMACH
NEOPLASMS, MALIGNANT, 4394
ULCER, 4394

FISSURE
ANUS
CROHNS DISEASE, 5194

FISTULA
ANUS
MORPHOLOGY, 4674
BILE DUCT
GALLBLADDER DISEASE, 5006
CHOLELITHIASIS
COMPLICATIONS, 4954*
SURGERY, 4954*
COLON
SIMULATION, 4675
GALLBLADDER
NEOPLASMS, MALIGNANT, 4994
GASTROINTESTINAL
ENDOSCOPY, 5122
HYPERALIMENTATION, 5122
LARGE INTESTINE
DIAGNOSIS, 4579
ELECTROLYTE, 4672
NUTRITION, 4672
SURGERY, 4672, 4673
THERAPY, 4579
SMALL INTESTINE
DIAGNOSIS, 4579
HYPERALIMENTATION, 4581
SURGERY, 4580, 4582, 4673
THERAPY, 4579
STOMACH
JEJUNUM, 5122
NEOPLASMS, BENIGN, 4431

FISTULAS
ANUS
CROHNS DISEASE, 5192

FLUORIDE
METABOLISM
CALCIUM, 3855
PHOSPHORUS, 3855

FOLIC ACID
CROHNS DISEASE
DRUG TREATMENT, 5195
DEFICIENCY
LIVER DISEASE, ALCOHOLIC, 4913
METABOLISM
SPRUE, TROPICAL, 4623

FOOD
FUNGICIDES
ABSORPTION, 3846
TRACE ELEMENT
ABSORPTION, 3846

FOOD ADDITIVES
INTESTINE
ABSORPTION, 3917
PATHOLOGY, 3917
LIVER
PATHOLOGY, 3917

FOREIGN BODIES
STOMACH
DRUG TREATMENT, 4417
ENZYMES, 4417

FRUCTOSE
INTOLERANCE
GENETIC FACTORS, 5106
IRON
ABSORPTION, 5091
METABOLISM
ENZYMES, 4149*
SMALL INTESTINE, 4149*

FUNGICIDES
FOOD
ABSORPTION, 3846
NUCLEIC ACIDS
SYNTHESIS, 4063
PROTEIN
SYNTHESIS, 4063

GALACTOSAMINE
HEPATITIS
SIMULATION, 4108
LIVER
NECROSIS, 4115

GALACTOSE
CIRRHOSIS
DIAGNOSIS, 4198*
HEPATITIS
DIAGNOSIS, 4198*
JAUNDICE, OBSTRUCTIVE
DIAGNOSIS, 4198*

GALLBLADDER
AMINO ACIDS
ABSORPTION, 3819*
ANOMALY, CONGENITAL, 5005
COMPLICATIONS, 4971
EPIDEMIOLOGY, 4971
SURGERY, 4970, 4971
BILE
AMYLASE, 4965
GLUCOSE, 4993
CHLORIDE
TRANSPORT, 3856
CIRCULATION
CHOLECYSTITIS, 4135
CYSTS
RUPTURE, 4979
TRAUMA, 4979
GASTRECTOMY
TECHNIQUES, 4401
HETEROTOPIA
RADIOLOGY, 4968
MOTILITY
ANOMALY, CONGENITAL, 4958
CALCIUM, 4736
CELIAC DISEASE, 4609*
CHOLELITHIASIS, 4944*
PANCREAS DISEASE, 3882
NEOPLASMS, BENIGN
DIAGNOSIS, 4963
NEOPLASMS, MALIGNANT
DIARRHEA, 4994
FISTULA, 4994
PERFORATION, 4994

GALLBLADDER (continued)
NERVOUS SYSTEM
CHOLECYSTITIS, 4135
PATHOLOGY
CALCULI, 4998
PRESSURE STUDIES
PENTAGASTRIN, 3869*
PROSTAGLANDIN, 3869*
RADIOLOGY
BILIARY DISEASE, 4992
TECHNIQUES, 4280
SUGAR
ABSORPTION, 3819*
TORSION
DIAGNOSIS, 4964
ETIOLOGY, 4964

GALLBLADDER DISEASE
AMINO ACIDS
METABOLISM, 4975
ANGIOGRAPHY, 4276
ENDOSCOPY, 4287
TECHNIQUES, 4287
BILE ACIDS
METABOLISM, 4125
CHOLECYSTOKININ
RADIOLOGY, 4286
COLITIS
ISCHEMIA, 4703
COMPLICATIONS
CHOLECYSTECTOMY, 4978
DRAINAGE, 4978
FISTULA
BILE DUCT, 5006
HYPERPLASIA
RADIOLOGY, 4275
RADIOLOGY
TECHNIQUES, 4213
SURGERY
TECHNIQUES, 4946*

GALLSTONE
CHOLESTEROL
ETIOLOGY, 4941*
ILEUS
DIAGNOSIS, 4996
SURGERY, 4996
INTESTINE
OBSTRUCTION, 4570, 4571

GARDNERS SYNDROME
RADIOLOGY, 4682
SURGERY
TECHNIQUES, 4679

GASTRECTOMY
SEE ALSO ANTRECTOMY
ESOPHAGUS
REFLUX, 4403
GASTRIN
METABOLISM, 3963
GASTROINTESTINAL
MOTILITY, 3909
IRON
KINETICS, 4406
JEJUNUM
LACTASE, 4407

GASTRECTOMY (continued)
SEQUELAE
DIGESTION, 4410
DRUG TREATMENT, 4404
REFLUX, 4403
STOMACH
NEOPLASMS, MALIGNANT, 4456
TECHNIQUES, 4405
DUMPING SYNDROME, 4401
GALLBLADDER, 4401
SEQUELAE, 4401

GASTRIN
ANTRUM
ACIDITY, 3966
CHEMICAL COMPOSITION, 3942*
NERVOUS CONTROL, 3936*
BLOOD, 5059
ANTRECTOMY, 3924*
ANTRUM, 3962
ATROPINE, 3965
BILE, 3988
BLOOD GROUP, 3969
CHEMICAL COMPOSITION, 3942*, 3973
CIRCADIAN RHYTHM, 3932*
DIETARY FACTORS, 3931*
FEEDING, 3932*
H2 RECEPTOR ANTAGONISTS, 3958
HISTAMINE, 3958
HYPERCALCEMIA, 3943*
PEPTIC ULCER, 4488*
PROSTAGLANDIN, 3967
TECHNIQUES, 4226
VAGOTOMY, 3924*
DUODENUM
CHEMICAL COMPOSITION, 3942*
ELECTROLYTE
ABSORPTION, 3839
GASTROINTESTINAL DISEASE
REVIEWS, 5024
GLUCOSE
ABSORPTION, 3839
HYPERCHLORHYDRIA, 4398, 4399
METABOLISM
GASTRECTOMY, 3963
PEPTIC ULCER
ANTICHOLINERGIC AGENTS, 4491*
H2 RECEPTOR ANTAGONISTS, 4491*
SECRETION
PATHOLOGY, 4471
PEPTIC ULCER, 4471
STOMACH DISEASE, 4471
STOMACH
ACID SECRETION, 3956, 3973
KINETICS, CELL, 3964
MORPHOLOGY, 3798, 3799, 3960
MOTILITY, 3888
PATHOLOGY, 3798
SECRETION, 3960, 3961, 3968
WATER
ABSORPTION, 3839

GASTRITIS
BLOOD
FIBRINOLYSIS, 4394
DRUG-INDUCED
CORTICOSTEROIDS, 3920*
DRUG TREATMENT, 4427

GASTRITIS (continued)
ENDOSCOPY, 4423
ETIOLOGY
DIETARY FACTORS, 4375*
SMOKING, 4375*
LACTASE DEHYDROGENASE
ISOENZYMES, 4428
SEQUELAE, 4430
SMALL INTESTINE
ELECTROLYTE, 5053
STOMACH
BLEEDING, 4421
PRECANCER, 4422

GASTRITIS, ATROPHIC
ANTRUM
EPIDEMIOLOGY, 4481
STOMACH
IMMUNOGLOBULINS, 4429
KINETICS, CELL, 4412

GASTRITIS, EROSIVE
BLEEDING, 4426
EPIDEMIOLOGY, 4382*
SURGERY, 4379*

GASTROENTERITIS
CHILDREN
ANTIBIOTICS, 5082
CIRRHOSIS
COMPLICATIONS, 4927
ESCHERICHIA COLI
EPIDEMIOLOGY, 5112
NEONATE, 5112
NEONATE
AMINO ACIDS, 5087
LIVER FUNCTION TESTS, 5087

GASTROINTESTINAL
ABSORPTION
HORMONE EFFECTS ON, 3877
BIOPOTENTIALS
TECHNIQUES, 5019*
BLEEDING, 5036
ASPIRIN, 4437, 4438
CIRRHOSIS, 4264
CROHNS DISEASE, 5187
DIAGNOSIS, 5081
DRUG-INDUCED, 4374*, 4421, 4437
DRUG TREATMENT, 5060
ENDOSCOPY, 4206, 4208, 4264, 5130
HEMATEMESIS, 5126
LEUKEMIA, 5029
MELENA, 5126
PANCREAS DISEASE, 4729
RADIOLOGY, 4207
SIMULATION, 4438
SURGERY, 5035, 5061, 5062
TELANGIECTASIA, 5123
THERAPY, 5130
VASOPRESSIN, 5015*
CARCINOIDS
DIAGNOSIS, 5120
ETIOLOGY, 5120
COMPLICATIONS
GASTROSTOMY, 4416
INTUBATION, 4416
ENDOCRINE SYSTEM
NEOPLASMS, 5114

GASTROINTESTINAL (continued)
ENDOSCOPY
CHILDREN, 4291
CIRCULATION, 4211
COMPUTERS, 4205
RESPIRATORY SYSTEM, 4211
REVIEWS, 4203
TECHNIQUES, 4221, 4291
ENZYMES
ANTIBACTERIALS, 3991
ANTIBIOTICS, 3991
FISTULA
ENDOSCOPY, 5122
HYPERALIMENTATION, 5122
HORMONE
CIRCULATION, 3872*
REVIEWS, 5039
MORPHOLOGY
ABSORPTION, 3820
DIGESTION, 3820
IRRADIATION, 3804
MOTILITY
BIOPOTENTIALS, 3878
CHOLECYSTECTOMY, 3879
ELECTRICAL CONTROL, 3879
GASTRECTOMY, 3909
HORMONE EFFECTS ON, 3877
PANCREATITIS, 4165*
TECHNIQUES, 3880
VOMITING, 3878
NEOPLASMS
ENDOCRINE SYSTEM, 5116
IMMUNOLOGY, 5028
SEQUELAE, 4430
NEOPLASMS, MALIGNANT
ALCOHOLISM, 5094
ASBESTOS, 4176
ENDOSCOPY, 4289
SURGERY, 5065
PATHOLOGY
URTICARIA PIGMENTOSA, 4395
PERFORATION
ETIOLOGY, 5104
NEONATE, 5104
POLYPS, 5066
PROTEIN-LOSING ENTEROPATHY, 5043
RADIOLOGY, 4202
SECRETION
DRUG EFFECTS ON, 3918
GASTROSTOMY, 4416
INTUBATION, 4416
SURGERY
GASTROSTOMY, 5054

GASTROINTESTINAL DISEASE
CHILDREN
ENDOSCOPY, 4201
DUODENUM
BIOPSY, 5051
GASTRIN
REVIEWS, 5024
LYMPHATICS, 5055
OXALATE
ABSORPTION, 5026
PROTEIN
HYDROLYSIS, 5027
PSYCHOLOGICAL FACTORS, 5056, 5057
TRANQUILIZERS, 5077

GASTROINTESTINAL DISEASE (continued)
URINE
OXALATE, 5041

GASTROINTESTINAL TRACT
BILE ACIDS
ABSORPTION, 3859
BLEEDING
ANGIOGRAPHY, 5022
DRUG ABSORPTION, 3821
ENDOCRINE SYSTEM
CARCINOIDS, 4284
MORPHOLOGY, 5117
NEOPLASMS, 4283
PATHOLOGY, 5117
IRRADIATION
AGE FACTORS, 4164*
LYMPHATICS
NEOPLASMS, MALIGNANT, 5048
NEOPLASMS
PATHOLOGY, 5118
NERVOUS SYSTEM, 4175
PERSORPTION, 3813*
PROSTAGLANDIN
ISOLATION, 3916
SEROTONIN
IRRADIATION, 4164*

GASTROSTOMY
ESOPHAGUS
COMPLICATIONS, 4365
GASTROINTESTINAL
COMPLICATIONS, 4416
SECRETION, 4416
SURGERY, 5054
TECHNIQUES
REVIEWS, 4409

GENETIC FACTORS
ANTITRYPSIN, ALPHA
DEFICIENCY, 4838
CHOLELITHIASIS
EPIDEMIOLOGY, 4960
FRUCTOSE
INTOLERANCE, 5106
MOUTH
NEOPLASMS, BENIGN, 5021
PEPTIC ULCER, 4493*
SHIGELLOSIS
IMMUNITY, 5075

GILBERTS DISEASE
BILIRUBIN
METABOLISM, 4191*
DIAGNOSIS
BILIRUBIN, 4191*

GLUCAGON
ELECTROLYTE
ABSORPTION, 3815*
GLUCOSE
ABSORPTION, 3815*

GLUCONEOGENESIS
LIVER
CORTICOSTEROIDS, 4077
HYPOXIA, 4077
NUCLEOTIDES, 4080

GLUCOSE
ABSORPTION
CELIAC DISEASE, 4605*
DIETARY FACTORS, 4153*
GASTRIN, 3839
GLUCAGON, 3815*
JEJUNUM, 3815*
SMALL INTESTINE, 3839
CARBOHYDRATE
DIGESTION, 3814
DIGESTION
DIETARY FACTORS, 4153*
GALLBLADDER
BILE, 4993
TRANSPORT
LIVER, 4093

GLUTEN
CELIAC DISEASE
PATHOLOGY, 4631
PROTEIN, 4629
INTOLERANCE
CELIAC DISEASE, 4602*
CHILDREN, 4602*
XYLOSE, 4602*
MALABSORPTION
REVIEWS, 4613
REGIONAL ENTERITIS
ETIOLOGY, 5159*
SPRUE
COMPLICATIONS, 4624

GLYCOGEN
LIVER
CHEMICAL COMPOSITION, 4079
SALIVARY GLANDS
CATECHOLAMINES, 3912
SYNTHESIS
VITAMIN D, 4112

GLYCOLYSIS
SMALL INTESTINE
DIETARY FACTORS, 4161
SUGAR, 4161

GLYCOPROTEINS
SMALL INTESTINE
CHOLERA, 5074
STOMACH
CARBENOXALONE, 4503*
NEOPLASMS, MALIGNANT, 4443
SECRETION, 4443
ULCERATIVE COLITIS, 4714
ULTRASTRUCTURE, 3788*
SYNTHESIS
INFLAMMATION, 4085
LIVER, 4085

GNOTOBIOSIS
BILE ACIDS
METABOLISM, 4167*

GROWTH
HORMONE
PANCREAS, 3785*
SMALL INTESTINE, 3785*
STOMACH, 3785*

GROWTH FACTOR
LIVER
PROTEIN, 4019
H2 RECEPTOR ANTAGONISTS
BLOOD
GASTRIN, 3958
HISTAMINE
METABOLISM, 4502*
PANCREAS
SECRETION, 4483*
PEPTIC ULCER
ACID SECRETION, 4491*
GASTRIN, 4491*
STOMACH
ACID SECRETION, 3972
SECRETION, 3922*, 4483*
ZOLLINGER-ELLISON SYNDROME, 5025

HAMARTOMA
ESOPHAGUS
POLYPS, 4343

HEIDENHAIN POUCH
STOMACH
ACID SECRETION, 3946*

HELMINTHIASIS
BIOCHEMISTRY, 5152
EPIDEMIOLOGY, 5135, 5136, 5141
REVIEWS, 5134
TRANSMISSION
ENVIRONMENTAL FACTORS, 5151
INTESTINE, 5151

HEMATEMESIS
GASTROINTESTINAL
BLEEDING, 5126
STOMACH
INTUSSUSCEPTION, 4475
HEMATOMA
DUODENUM
SURGERY, 4568
SMALL INTESTINE, 4567
OBSTRUCTION, 4569

HEMODIALYSIS
INTESTINE
ABSORPTION, 4190*
SEQUELAE
LIVER DISEASE, 4791*

HEMORRHAGE
SEE BLEEDING

HEMORRHOIDS
DIETARY FACTORS
FIBER, 5089

HEPATECTOMY
BLOOD
PROTEIN, 4016
COMPLICATIONS, 4831
LIVER
REGENERATION, 4015*, 4021, 4034
ULTRASTRUCTURE, 4143
LIVER DISEASE, 4833
NUCLEIC ACIDS
SYNTHESIS, 4015*, 4020
TECHNIQUES, 4831

HEPATITIS
DIAGNOSIS
GALACTOSE, 4198*
STIMULATION
DRUG TREATMENT, 4108
ENDOCRINE DISEASE, 4109
GALACTOSAMINE, 4108

HEPATITIS, CHRONIC, 4921*
ANTIGEN, AUSTRALIA
IMMUNITY, 4901*
SMALL INTESTINE, 4897
AUTOIMMUNITY, 4796*
CIRRHOSIS, BILIARY, 4796*
BLOOD
CLOTTING, 4899*
IMMUNOGLOBULINS, 4903*
PHOSPHOLIPID, 4923*
CORTICOSTEROIDS
LYMPHOCYTE, 4871*
DIAGNOSIS, 4266
DISEASES ASSOCIATED WITH, 4900*
DRUG TREATMENT
COMPLICATIONS, 4907
CORTICOSTEROIDS, 4910
IMMUNOSUPPRESSION, 4906*
DUODENUM
PATHOLOGY, 5099
ENZYMES
BIOPSY, 4265
ETIOLOGY
AUTOIMMUNITY, 4900*
FIBRINOLYSIS
BLOOD, 4899*
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4876*, 4879
IMMUNOGLOBULINS, 4801, 4802
IMMUNOGLUBULINS
FAMILIAL FACTORS, 4903*
IMMUNOLOGY, 4900*
ETIOLOGY, 4925*
IMMUNOSUPRESSION, 4910
IMMUNOSUPPRESSION
CHILDREN, 4905*
LIVER
ULTRASTRUCTURE, 4908
SCANNING, SCINTILLATION, 4911
SIMULATION
LYSOSOMES, 4110
SMALL INTESTINE
ELECTROLYTE, 5053
ULTRASTRUCTURE, 4897
STOMACH
PATHOLOGY, 5099
SURGERY
TECHNIQUES, 4909

HEPATITIS, NONVIRAL
AFLATOXIN, 4861*
CHOLESTASIS
DRUG-INDUCED, 4862*
DRUG-INDUCED
ANESTHETICS, 4863
ANTIBACTERIALS, 4858*, 4859*, 4864
RISK FACTORS, 4859*
TRANSFUSION, 4864
SYPHILIS
DIAGNOSIS, 4860*

HEPATITIS, VIRAL
ANGIOGRAPHY
CIRRHOSIS, 4881
ANTIGEN
EPIDEMIOLOGY, 4878
RADIOIMMUNOASSAY, 4878
ANTIGEN, AUSTRALIA
CARRIER STATE, 4898
CHILDREN, 4876*
CORTICOSTEROIDS, 4870*, 4882
ELECTROPHORESIS, 4896
EPIDEMIOLOGY, 4877, 4882, 4893
FECES, 4896
HEPATITIS, CHRONIC, 4876*, 4879
IMMUNOLOGY, 4873*, 4880
RADIOIMMUNOASSAY, 4896
SMALL INTESTINE, 4897
STATISTICAL STUDY, 4877, 4893
URINE, 4896
ATROPHY
PERFUSION, 4895
AUTOIMMUNITY, 4796*
BLOOD
CLOTTING, 4885, 4886, 4887
HISTAMINE, 4891
IMMUNOGLOBULINS, 4874*
VITAMIN B12, 4883
CHILDREN
CORTICOSTEROIDS, 4870*
CORTICOSTEROIDS
LYMPHOCYTE, 4871*
DIAGNOSIS
PATHOLOGY, 4285
DRUG TREATMENT
CHILDREN, 4890
FIBRINOLYSIS, 4884, 4885
HYPERLIPOPROTEINEMIA, 4888
ISOENZYMES
LACTATE DEHYDROGENASE, 4894
JAUNDICE
DIAGNOSIS, 4268
KIDNEY
TRANSPLANTATION, 4786*
LIVER
ULTRASTRUCTURE, 4892
STOMACH
MORPHOLOGY, 4889
SURVIVAL
BIOPSY, 4875*
COMPLICATIONS, 4875*
NECROSIS, 4875*
TRANSFUSION
TRANSMISSION, 4872*
VITAMIN B12
RADIOISOTOPES, 4883

HERNIA
CHILDREN
SURGERY, 4584
DIAPHRAGM
DIAGNOSIS, 4326
SURGERY, 4326
ESOPHAGUS
REFLUX, 4314
SIMULATION, 4314
SPHINCTER, 4318
STRANGULATION, 4362
SURGERY, 4314

HERNIA (continued)
SMALL INTESTINE
RUPTURE, 4583
ULCERATIVE COLITIS
COMPLICATIONS, 4710*

HERNIA, HIATUS
CHILDREN
RADIOLOGY, 4324
COMPLICATIONS, 4325
DIETARY FACTORS
FIBER, 5089
DISEASES ASSOCIATED WITH
SURGERY, 4331
ESOPHAGUS
REFLUX, 4363
ULCER, 4313
NEONATE
RADIOLOGY, 4324
OBESITY, 4323
RADIOLOGY, 4315

HETEROTOPIA
GALLBLADDER
RADIOLOGY, 4968
INTESTINE
SCANNING, SCINTILLATION, 4210
PANCREAS
DUODENUM, 4733
WHIPPLES DISEASE, 4625
STOMACH
ENTEROCOLITIS, 4450
NEOPLASMS, MALIGNANT, 4574
SURGERY, 4450

HIRSCHSPRUNGS DISEASE
BILE ACIDS
EXCRETION, 4656*
DIAGNOSIS
REVIEWS, 4708
ENTEROCOLITIS, 4681
ETIOLOGY
REVIEWS, 4708
NERVOUS SYSTEM
COMPLICATIONS, 4662
PATHOLOGY
REVIEWS, 4708
RADIOLOGY
NEONATE, 4662

HISTAMINE
BLOOD
GASTRIN, 3958
HEPATITIS, VIRAL, 4891
LIVER, 4098
PANCREATITIS, 4756
HYPERCHLORHYDRIA
ACID SECRETION, 4507*
METABOLISM
H2 RECEPTOR ANTAGONISTS, 4502*
INTESTINE, 4159
LIVER, 4098
PEPTIC ULCER, 4502*
VITAMIN B6, 4159
PEPTIC ULCER
ACID SECRETION, 4504*, 4507*
STOMACH
ACID SECRETION, 3972, 4492*
VASODILATORS, 3934*

HODGKINS DISEASE
DUODENUM
BLEEDING, 4557
PERFORATION, 4557
ENDOSCOPY
LYMPHATICS, 4261

HORMONE
BILIARY
EXCRETION, 3852
GASTROINTESTINAL
CIRCULATION, 3872*
REVIEWS, 5039
GROWTH
PANCREAS, 3785*
SMALL INTESTINE, 3785*
STOMACH, 3785*
LIVER
ULTRASTRUCTURE, 3794*
METABOLISM
LIVER, 3852, 4099
THYROID
CROHNS DISEASE, 5160*, 5161*
ULCERATIVE COLITIS, 5160*, 5161*

HORMONE CONTROL
LIVER
POTASSIUM, 3995*
PANCREAS
SECRETION, 3952
PEPSIN
SECRETION, 3944*, 3951, 3954
STOMACH
ACID SECRETION, 3944*, 3945*, 3951, 3954, 4396
SECRETION, 3952, 4494*

HORMONE EFFECTS ON
ESOPHAGUS
SPHINCTER, 4318
GASTROINTESTINAL
ABSORPTION, 3877
MOTILITY, 3877
ION TRANSPORT
INTESTINE, 3816*
JEJUNUM
ABSORPTION, 3808*
ADENYL CYCLASE, 3817*
ION TRANSPORT, 3808*
PEPSIN
SECRETION, 3887
PROTEIN
METABOLISM, 4089
SYNTHESIS, 4087
STOMACH
MOTILITY, 3887
SECRETION, 3920*, 3953
ULCER
STRESS, 4384*

HORMONES
DUODENUM
BIOPOTENTIALS, 3831

HYDROCARBONS, HALOGENATED
METABOLISM
LIVER INJURY, 4062

HYDROLYSIS
PROTEIN
GASTROINTESTINAL DISEASE, 5027
STOMACH DISEASE, 5027

HYPERALIMENTATION
GASTROINTESTINAL
FISTULA, 5122
SMALL INTESTINE
FISTULA, 4581

HYPERBILIRUBINEMIA
ALBUMIN, 4855
ANTIBIOTICS, 4855
ASPIRIN
METABOLISM, 4854*
BILIRUBIN
METABOLISM, 4854*
PHOTOTHERAPY, 4855

HYPERCALCEMIA
BLOOD
GASTRIN, 3943*
STOMACH
SECRETION, 3943*

HYPERCAPNIA
STOMACH
PATHOLOGY, 4366*

HYPERCHLORHYDRIA
SEE ALSO ACID SECRETION
ACID SECRETION
CALCIUM, 4507*
HISTAMINE, 4507*
GASTRIN, 4398, 4399

HYPERLIPEMIA
COMPLICATIONS
PANCREATITIS, 4757

HYPERLIPOPROTEINEMIA
HEPATITIS, VIRAL, 4888
LIPIDS
LIVER, 4797*
PROTEINS
LIVER, 4797*

HYPERPLASIA
GALLBLADDER DISEASE
RADIOLOGY, 4275
SMALL INTESTINE
LYMPHATICS, 4444
STOMACH
LYMPHATICS, 4444

HYPERTENSION
LIVER FUNCTION TESTS
AGE FACTORS, 4842
RECTUM
BIOPOTENTIALS, 4663

HYPNOSIS
STOMACH
ACID SECRETION, 3941*

HYPOCHLORHYDRIA
SEE ALSO ACID SECRETION

HYPOCHLORHYDRIA (continued)
LACTASE DEHYDROGENASE
ISOENZYMES, 4428
ZOLLINGER-ELLISON SYNDROME
DIARRHEA, 5121

HYPOLIPEMIC AGENTS
LIVER
DRUG METABOLISM, 3854

HYPOPLASIA
BILE DUCT
ANTITRYPSIN, ALPHA, 4838

HYPOXIA
ENTEROCOLITIS
STIMULATION, 5017*
LIVER
GLUCONEOGENESIS, 4077
PANCREAS
SECRETION, 3978*

ICTERUS
SEE JAUNDICE

ILEOSTOMY
DRAINAGE
DIETARY FACTORS, 4709*

ILEUM
SEE ALSO SMALL INTESTINE
ABSORPTION
CELIAC DISEASE, 4605*
CROHNS DISEASE
RECURRENCE, 5189
MAGNESIUM
SEROTONIN, 3875*
MOTILITY
ACETYLCHOLINE, 3870*
DRUG EFFECTS ON, 3890
ELECTRICAL CONTROL, 3870*
NARCOTICS, 3890, 3891
SEROTONIN, 3875*
PERISTALSIS
NERVOUS CONTROL, 3897
POLYPS
LYMPHATICS, 4680
SULFATE
ABSORPTION, 3848

ILEUS
GALLSTONE
DIAGNOSIS, 4996
SURGERY, 4996
SMALL INTESTINE
MOTILITY, 3906
THERAPY, 4572

IMMUNITY
CIRRHOSIS
PORTACAVAL SHUNT, 4924*
COLON
NEOPLASMS, 4690
HEPATITIS, CHRONIC
ANTIGEN, AUSTRALIA, 4901*
PROTEIN
CELIAC DISEASE, 4607*
SMALL INTESTINE, 4607*
SHIGELLOSIS
GENETIC FACTORS, 5075

IMMUNOGLOBULINS
BLOOD
HEPATITIS, CHRONIC, 4903*
HEPATITIS, VIRAL, 4874*
CELIAC DISEASE
COMPLICATIONS, 4643
REVIEWS, 4640
CIRRHOSIS, 4801, 4802, 4921*
DIAGNOSIS, 4914
ENTERITIS
VIRUS, 5014*
HEPATITIS, CHRONIC, 4801, 4802
FAMILIAL FACTORS, 4903*
LARGE INTESTINE
CROHNS DISEASE, 5166*
LIVER
NEOPLASMS, MALIGNANT, 4801
LIVER DISEASE, ALCOHOLIC
DIAGNOSIS, 4914
REGIONAL ENTERITIS, 5159*
SALMONELLOSIS
INFECTION, 5107
SMALL INTESTINE
CHOLERA, 5074
CROHNS DISEASE, 5166*
STOMACH
CIRRHOSIS, 4429
GASTRITIS, ATROPHIC, 4429

IMMUNOLOGY
CELIAC DISEASE
CHILDREN, 4632
REVIEWS, 4640
CIRRHOSIS, 5184
ETIOLOGY, 4925*
PORTACAVAL SHUNT, 4924*
COLON
NEOPLASMS, MALIGNANT, 4648*, 4687
CROHNS DISEASE, 5170*
DYSENTERY
CHILDREN, 5073
GASTROINTESTINAL
NEOPLASMS, 5028
HEPATITIS, CHRONIC, 4900*
ETIOLOGY, 4925*
IMMUNOSUPRESSION, 4910
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4873*, 4880
LEUKOCYTES
ULCERATIVE COLITIS, 4715
PARASITES AND PARASITIC DISEASE
DIAGNOSIS, 4282
PROTEIN
INTOLERANCE, 4621
REGIONAL ENTERITIS, 5184
SALMONELLOSIS
INFECTION, 5107
ULCERATIVE COLITIS, 5184
WHIPPLES DISEASE, 4611*
ZINC, 5129

IMMUNOSUPPRESSION
CROHNS DISEASE
DRUG TREATMENT, 5188
HEPATITIS, CHRONIC
CHILDREN, 4905*

IMMUNOSUPPRESSION (continued)
HEPATITIS, CHRONIC (continued)
DRUG TREATMENT, 4906*
IMMUNOLOGY, 4910
IRRADIATION
TRAUMA, 4180
KIDNEY
TRANSPLANTATION, 4498*
LIVER
TRANSPLANTATION, 4027

INCONTINENCE
ANORECTUM
PRESSURE STUDIES, 4651*

INDOCYANINE GREEN
BINDING
AGE FACTORS, 3998*
LIVER, 3998*
LIVER FUNCTION TESTS, 4260
METABOLISM
LIVER, 3807*

INFECTION
COLON
NEOPLASMS, MALIGNANT, 4694
DIARRHEA
CHILDREN, 5020
DISACCHARIDASE
DEFICIENCY, 5098
INTESTINE
DISEASES ASSOCIATED WITH, 5032
INTERFERON, 5032
THERAPY, 5040
PHOSPHATASE, ALKALINE
DEFICIENCY, 5098
PROTOZOA
EPIDEMIOLOGY, 5135
SALMONELLOSIS
IMMUNOGLOBULINS, 5107
IMMUNOLOGY, 5107

INFECTION, BACTERIA
BILE
SYNTHESIS, 4137
BILIARY
ANTIBIOTICS, 4986
ENTEROCOLITIS, 5033
ESOPHAGUS
DIAGNOSIS, 4311
INTESTINE
ETIOLOGY, 5076
LIVER
BRUCELLOSIS, 4792*
LEPROSY, 4792*
TUBERCULOSIS, 4792*
LIVER COMA
CIRRHOSIS, 4912

INFECTION, VIRUS
INTESTINE
MORPHOLOGY, 3792*

INFLAMMATION
ALBUMIN
SYNTHESIS, 4085
BILE DUCT
DIAGNOSIS, 4974

INFLAMMATION (continued)
ESOPHAGUS
PSEUDOTUMOR, 4364
GLYCOPROTEINS
SYNTHESIS, 4085
MEDIASTINUM
ESOPHAGUS DISEASE, 4347

INFLAMMATORY BOWEL DISEASE
AMEBIASIS
COLITIS, 5131*
CHILDREN
SURGERY, 4720
DRUG TREATMENT
DRUG TOXICITY, 4182
MAGNESIUM
METABOLISM, 5191
SURGERY
SEQUELAE, 4719

INTEGUMENTARY SYSTEM
TEMPERATURE
CHOLECYSTITIS, 4537
PEPTIC ULCER, 4537

INTERFERON
INTESTINE
INFECTION, 5032

INTESTINE
SEE ALSO SMALL INTESTINE
ABSORPTION
DRUG EFFECTS ON, 3833
FOOD ADDITIVES, 3917
HEMODIALYSIS, 4190*
MORPHOLOGY, 3800
NERVOUS SYSTEM, 3827
XYLOSE, 4231
AMINO ACIDS
ABSORPTION, 3810*
BILE ACIDS
JAUNDICE OBSTRUCTIVE, 4130
CALCIUM
TRANSPORT, 3853
DRUG ABSORPTION, 3822
DRUGS
SECRETION, 3822
ELECTROLYTE
ABSORPTION, 3830
FATTY ACIDS
ABSORPTION, 3850
HELMINTHIASIS
TRANSMISSION, 5151
HETEROTOPIA
SCANNING, SCINTILLATION, 4210
HISTAMINE
METABOLISM, 4159
INFECTION
DISEASES ASSOCIATED WITH, 5032
INTERFERON, 5032
THERAPY, 5040
INFECTION, BACTERIA
ETIOLOGY, 5076
ION TRANSPORT
HORMONE EFFECTS ON, 3816*
IRON
ABSORPTION, 3828
IRRADIATION
TRAUMA, 4180, 4185

INTESTINE (continued)
KINETICS, CELL
AUTORADIOGRAPHY, 3802
LEUKEMIA
INFILTRATION, 4556
MALABSORPTION
ALCOHOLISM, 3835
METABOLISM
VITAMIN D, 4163
METAPLASIA
ENDOSCOPY, 4192*
MICROORGANISMS
MORPHOLOGY, 5157
MITOSIS
CIRCADIAN RHYTHM, 4179
RADIATION, 4179
MORPHOLOGY
INFECTION, VIRUS, 3792*
PORTACAVAL SHUNT, 4038
MOTILITY
ANTIBIOTICS, 5013*
ELECTROPHYSIOLOGY, 3895
NERVOUS SYSTEM, 3892
TECHNIQUES, 3893
NEOPLASMS, MALIGNANT
ANTIGEN, CARCINOEMBRYONIC, 4178
NUCLEIC ACIDS
METABOLISM, 4163
OBSTRUCTION
GALLSTONE, 4570, 4571
SURGERY, 4571
PATHOLOGY
CROHNS DISEASE, 5190
FOOD ADDITIVES, 3917
UROGENITAL SYSTEM, 4660
PERMEABILITY
LAXATIVES, 3833
PROTOZOA
EPIDEMIOLOGY, 5140
STOMACH
ACID SECRETION, 3946*
SURFACTANTS
ABSORPTION, 3823
SURGERY
TECHNIQUES, 3898
TUBERCULOSIS, 4652*
ULTRASTRUCTURE
EMBRYOLOGY, 3793*

INTOLERANCE
DISACCHARIDASE
DIARRHEA, 5045
NEONATE, 5045
FRUCTOSE
GENETIC FACTORS, 5106
GLUTEN
CELIAC DISEASE, 4602*
CHILDREN, 4602*
XYLOSE, 4602*
PROTEIN
IMMUNOLOGY, 4621
SMALL INTESTINE, 4606*

INTUBATION
GASTROINTESTINAL
COMPLICATIONS, 4416
SECRETION, 4416
STOMACH DISEASE
TRAUMA, 4436

INTUSSUSCEPTION
COLON, 4691
SMALL INTESTINE
RADIOLOGY, 4586
SURGERY, 4585
STOMACH
HEMATEMESIS, 4475

IODINE
THYROID
CROHNS DISEASE, 5165*
ULCERATIVE COLITIS, 5165*

ION TRANSPORT
ANTRUM
REVIEWS, 3842
INTESTINE
HORMONE EFFECTS ON, 3816*
JEJUNUM
HORMONE EFFECTS ON, 3808*
LIVER
CARCINOGENS, 4148
PHENOBARBITAL, 4148
SMALL INTESTINE
DIURETICS, 4152*
DRUG EFFECTS ON, 4152*
STOMACH
ACID SECRETION, 3949

IRON
ABSORPTION
DUODENUM, 3851
FRUCTOSE, 5091
INTESTINE, 3828
NEONATE, 3828
PANCREATECTOMY, 5059
BINDING
STOMACH, 3948
CROHNS DISEASE
DRUG TREATMENT, 5195
DEFICIENCY
STOMACH, 4380*
KINETICS
GASTRECTOMY, 4406
STOMACH
SECRETION, 3948

IRRADIATION
GASTROINTESTINAL
MORPHOLOGY, 3804
GASTROINTESTINAL TRACT
AGE FACTORS, 4164*
SEROTONIN, 4164*
INTESTINE
TRAUMA, 4185
LIVER
ULTRASTRUCTURE, 3790*
STOMACH
ADENYL CYCLASE, 3970
PHOSPHODIESTERASE, 3970
TRAUMA
ENDOTOXIN, 4180
IMMUNOSUPRESSION, 4180
INTESTINE, 4180
LIVER, 4180
PREVENTION, 4185

IRRITABLE COLON
DIAGNOSIS, 4700
THERAPY, 4700

ISCHEMIA
COLITIS
COMPLICATIONS, 4703
GALLBLADDER DISEASE, 4703
PERFORATION, 4703
COLON
NEOPLASMS, MALIGNANT, 4695
LIVER, 4037
ENZYMES, 4036
SEQUELAE, 4035
STIMULATION, 4102
STOMACH
BIOPOTENTIALS, 3861*
PATHOLOGY, 4386*

ISOENZYMES
LACTATE DEHYDROGENASE
GASTRITIS, 4428
HEPATITIS, VIRAL, 4894
HYPOCHLORHYDRIA, 4428
LIVER INJURY
CARBON TETRACHLORIDE, 4061

JAUNDICE
DIAGNOSIS, 4267
ETIOLOGY
ANTIBACTERIALS, 4846
HEPATITIS, VIRAL
DIAGNOSIS, 4268
SURGERY
SEQUELAE, 4981

JAUNDICE, OBSTRUCTIVE
ANGIOGRAPHY, 4789*
BILE ACIDS
BLOOD, 4130
INTESTINE, 4130
CHOLECYSTITIS
COMPLICATIONS, 4976
DIAGNOSIS, 4268
GALACTOSE, 4198*
NEOPLASMS, 5069
LIVER
CIRCULATION, 4987
SEQUELAE
SURGERY, 4991

JEJUNUM
SEE ALSO SMALL INTESTINE
ABSORPTION
HORMONE EFFECTS ON, 3808*
ADENYL CYCLASE
HORMONE EFFECTS ON, 3817*
BIOPOTENTIALS
ANGIOTENSIN, 3884
BIOPSY
COMPLICATIONS, 4589
CROHNS DISEASE, 5162*
PERFORATION, 4589
ULCERATIVE COLITIS, 5162*
ELECTROLYTE
ABSORPTION, 3815*
ENTEROKINASE
MALABSORPTION, 4612*

JEJUNUM (continued)
GLUCOSE
ABSORPTION, 3815*
ION TRANSPORT
HORMONE EFFECTS ON, 3808*
KINETICS, CELL
CELIAC DISEASE, 4635
LACTASE
GASTRECTOMY, 4407
MOTILITY
ANGIOTENSIN, 3884
NEOPLASMS
PERFORATION, 4559
PATHOLOGY
CELIAC DISEASE, 4635
WHIPPLES DISEASE, 4625
PEPTIC ULCER
VAGOTOMY, 4522
STOMACH
FISTULA, 5122
ULTRASTRUCTURE
CELIAC DISEASE, 4633
WATER
ABSORPTION, 3815*

KIDNEY
CIRCULATION
LIVER DISEASE, 4103
COLON
NEOPLASMS, MALIGNANT, 4698
NEOPLASMS, BENIGN
LIVER DISEASE, 4848
TRANSPLANTATION
COMPLICATIONS, 4684
HEPATITIS, VIRAL, 4786*
IMMUNOSUPPRESSION, 4498*
PANCREATITIS, 4779
PEPTIC ULCER, 4498*
SEQUELAE, 4498*, 4791*

KIDNEY DISEASE
VITAMIN D
ABSORPTION, 5067
METABOLISM, 5067

KINETICS
COPPER
WILSONS DISEASE, 4782*
DRUG METABOLISM
LIVER, 4056
IRON
GASTRECTOMY, 4406
PARASITES AND PARASITIC DISEASE, 4170
SODIUM
METABOLISM, 3809*

KINETICS, CELL
BILE DUCT
CARBON TETRACHLORIDE, 4060
INTESTINE
AUTORADIOGRAPHY, 3802
JEJUNUM
CELIAC DISEASE, 4635
LIVER
AUTORADIOGRAPHY, 3802
NEOPLASMS, 5095
SMALL INTESTINE
ALKALOIDS, 4156

KINETICS, CELL (continued)
SMALL INTESTINE (continued)
AUTORADIOGRAPHY, 3801
CIRCADIAN RHYTHM, 4155*
NEONATE, 3801
PENTAGASTRIN, 4155*
SECRETIN, 4155*
STOMACH, 4412
GASTRIN, 3964
GASTRITIS, ATROPHIC, 4412
NEOPLASMS, MALIGNANT, 4412

KWASHIORKOR
THERAPY
DIET, 5016*

LACTASE
SEE ALSO DISACCHARIDASE
JEJUNUM
GASTRECTOMY, 4407

LACTATE DEHYDROGENASE
DIARRHEA
NEONATE, 5050
ISOENZYMES
GASTRITIS, 4428
HEPATITIS, VIRAL, 4894
HYPOCHLORHYDRIA, 4428
STOMACH
NEOPLASMS, MALIGNANT, 4428

LACTOSE
NITROGEN
DIGESTION, 4153*
PROTEIN
DIGESTION, 4153*

LACTOSE INTOLERANCE
DISEASES ASSOCIATED WITH
CELIAC DISEASE, 4603*
EPIDEMIOLOGY, 4644

LAMBLIASIS
SEE GIARDIASIS

LAPAROSCOPY
BILARY DISEASE, 4808
LIVER
NEOPLASMS, MALIGNANT, 4269
LIVER DISEASE, 4808

LARGE INTESTINE
SEE ALSO INTESTINE, APPENDIX, CECUM,
COLON, RECTUM, ANUS, SIGMOID
AMEBIASIS
ETIOLOGY, 5156
SURGERY, 5155
BLEEDING
DISEASES ASSOCIATED WITH, 4661
RADIOLOGY, 4212
SURGERY, 4661
DISEASES
CYTOLOGY, 4241
FISTULA
DIAGNOSIS, 4579
ELECTROLYTE, 4672
NUTRITION, 4672
SURGERY, 4672, 4673
THERAPY, 4579

LARGE INTESTINE (continued)
IMMUNOGLOBULINS
CROHNS DISEASE, 5166*
MICROORGANISMS
ULCERATIVE COLITIS, 4716
NEOPLASMS, MALIGNANT
COMPLICATIONS, 4693
CROHNS DISEASE, 5180
DIAGNOSIS, 4647*
METASTASES, 4647*
MUCOPOLYSACCHARIDE, 4685
OBSTRUCTION, 4693
PERFORATION, 4693
PREVENTION, 4678
OBSTRUCTION
ANTICOAGULANTS, 4669
DISEASES ASSOCIATED WITH, 4686
DRUG-INDUCED, 4669
DRUG TREATMENT, 4668
PATHOLOGY
CYTOMEGALOVIRUS, 5071
PNEUMATOSIS, 4706
POLYPS
SURGERY, 4721
ULCER
DISEASES ASSOCIATED WITH, 4665

LAXATIVES
BARIUM
ENEMA, 4243
DIARRHEA
DRUG-INDUCED, 5083
ELECTROLYTE
TRANSPORT, 3841
INTESTINE
PERMEABILITY, 3833
WATER
TRANSPORT, 3841

LEAD
LIVER
ULTRASTRUCTURE, 4064

LEPROSY
LIVER
INFECTION, BACTERIA, 4792*

LEUKEMIA
GASTROINTESTINAL
BLEEDING, 5029
INFILTRATION
INTESTINE, 4556

LEUKOCYTES
IMMUNOLOGY
ULCERATIVE COLITIS, 4715

LIPASE
BILE
LIPIDS, 3975*
BLOOD
PANCREATITIS, CHRONIC, 4253
LIPOPROTEINS
LIVER DISEASE, ALCOHOLIC, 4915
PANCREAS
NEOPLASMS, MALIGNANT, 4253
STOMACH
PEPTIC ULCER, 4505*

LIPIDS
ABSORPTION
BILE ACIDS, 3859
BILE
COENZYMES, 3975*
LIPASE, 3975*
BLOOD
CHOLELITHIASIS, 5001
PANCREATITIS, 4755
HYPERLIPOPROTEINEMIA
LIVER, 4797*
LIVER
CHEMICAL COMPOSITION, 4048
DIETARY FACTORS, 4047
VITAMIN A, 4048
METABOLISM
ALCOHOL, 4151*, 4154*
FATTY ACIDS, 3930*
SMALL INTESTINE, 4151*, 4154*
PEROXIDATION
LIVER, 3999*, 4052
LIVER INJURY, 4076
SECRETION
BILE, 4941*
PHENOBARBITAL, 3994*
STOMACH
DIAGNOSIS, 4413
ETIOLOGY, 4413
SYNTHESIS
DIETARY FACTORS, 4150*
LIVER, 4044
SMALL INTESTINE, 4150*

LIPOPROTEINS
LIPASE
LIVER DISEASE, ALCOHOLIC, 4915
SECRETION
LIVER DISEASE, ALCOHOLIC, 4915

LIVER
ABSCESS
AMEBIASIS, 5137, 5142, 5146, 5154
COMPLICATIONS, 5154
DIAGNOSIS, 5142
PREGNANCY, 5137
THERAPY, 5142
ALBUMIN
METABOLISM, 4092
SYNTHESIS, 4085
ALCOHOL
OXIDATION, 3996*, 3997*, 4049
PEROXIDATION, 4002*
AMEBIASIS
DIAGNOSIS, 5149
DRUG TREATMENT, 5149
AMINES
METABOLISM, 4072
AMINO ACIDS
METABOLISM, 3811*, 4078, 4090, 4091
AMMONIA
TOLERANCE TEST, 4101
ANGIOGRAPHY
CONTRAST MEDIA, 4106
BILE ACIDS
METABOLISM, 3807*
SYNTHESIS, 4128, 4129
BILE DUCT
LIVER DISEASE, 4799

LIVER (continued)
BILIRUBIN
TRANSPORT, 4117, 4119, 4121, 4123
BIOPSY
ANEMIA, SICKLE CELL, 4840
THALASSEMIA, 4840
BLOOD
HISTAMINE, 4098
CADMIUM
OCCUPATIONAL FACTORS, 4866
CARBOHYDRATE
METABOLISM, 3811*, 4007*
CARBON TETRACHLORIDE
PHENOBARBITAL, 4011*
CATALASE, 4002*
DEFICIENCY, 4114
CHOLESTEROL
SYNTHESIS, 4043
TRANSPORT, 4045
CIRCULATION, 4032
CIRRHOSIS, 4837, 4922*
JAUNDICE, OBSTRUCTIVE, 4987
MORPHOLOGY, 4033
PORTACAVAL SHUNT, 4040, 4933
CLOTTING
VITAMIN K, 4008*
COENZYMES
ULTRASTRUCTURE, 4113
COPPER
METABOLISM, 4082
CORTICOSTEROIDS
AGE FACTORS, 4096
METABOLISM, 4097
CYSTS
ANGIOGRAPHY, 4869
DIAGNOSIS, 4824
CYTOCHROMES
AGE FACTORS, 4083
SEX FACTORS, 4083
CYTOLOGY, 4298
DEHYDROGENASE
VITAMIN B1, 4086
DRUG METABOLISM
ALCOHOL, 4010*
ANTIBIOTICS, 4010*
CADMIUM, 4058
CHOLESTASIS, 4138
CYTOCHROMES, 4051
EMBRYOLOGY, 3787*
HYPOLIPEMIC AGENTS, 3854
KINETICS, 4056
MICROSOMES, 4052
ZINC, 4057
ECHINOCOCCOSIS
COMPLICATIONS, 5139
DIAGNOSIS, 5148
SURGERY, 5148
ENDOTOXIN
DETOXIFICATION, 4066
ENZYMES
PERFUSION, 4140
PHENOBARBITAL, 4067
FATTY ACIDS
DIABETES, 4844
FATTY LIVER, 4844
STARVATION, 4046
SYNTHESIS, 4055

LIVER (continued)
FATTY LIVER
CATALASE, 4787*
FIBRINOLYSIS, 4008*
GLUCONEOGENESIS
CORTICOSTEROIDS, 4077
HYPOXIA, 4077
NUCLEOTIDES, 4080
GLUCOSE
TRANSPORT, 4093
GLYCOGEN
CHEMICAL COMPOSITION, 4079
GLYCOPROTEINS
SYNTHESIS, 4085
GROWTH FACTOR
PROTEIN, 4019
HEPATECTOMY
REGENERATION, 4034
HISTAMINE
METABOLISM, 4098
HORMONE
METABOLISM, 3852, 4099
HYPERLIPOPROTEINEMIA
LIPIDS, 4797*
PROTEINS, 4797*
INDOCYANINE GREEN
BINDING, 3998*
METABOLISM, 3807*
INFECTION, BACTERIA
BRUCELLOSIS, 4792*
LEPROSY, 4792*
TUBERCULOSIS, 4792*
ION TRANSPORT
CARCINOGENS, 4148
PHENOBARBITAL, 4148
IRRADIATION
TRAUMA, 4180
ISCHEMIA, 4037
ENZYMES, 4036
SEQUELAE, 4035
SIMULATION, 4102
KINETICS, CELL
AUTORADIOGRAPHY, 3802
LIPIDS
CHEMICAL COMPOSITION, 4048
DIETARY FACTORS, 4047
PEROXIDATION, 3999*, 4052
SYNTHESIS, 4044
VITAMIN A, 4048
LYMPHATICS
ANGIOGRAPHY, 4817
STASIS, 4104
LYSOSOMES
PHOSPHATASE, ACID, 4041
MARKER STUDIES
CIRCULATION, 4816
METASTASES, 4198*
ENZYMES, 4830
LIVER FUNCTION TESTS, 4830
MICROSOMES
ENZYMES, 4084
MITOCHONDRIA
ULTRASTRUCTURE, 4054
MITOSIS
CARCINOGENS, 4034
EPINEPHRINE, 4144
STRESS, 4144
SURGERY, 4141

LIVER (continued)
MORPHOLOGY
BILE DUCT, 4126
BLEEDING, 4145
CARBON TETRACHLORIDE, 4073
PORTACAVAL SHUNT, 4038
STARVATION, 4851
TRANSFORMATION, 3786*
NECROSIS
BLEEDING, 4937
DIETARY FACTORS, 4071
FIBRINOLYSIS, 4886
GALACTOSAMINE, 4115
PREVENTION, 4071
TRACE ELEMENTS, 4071
NEOPLASMS
DIAGNOSIS, 4824
FETOPROTEIN, ALPHA, 4821
SCANNING, SCINTILLATION, 4821
SURGERY, 4827
NEOPLASMS, BENIGN
ANGIOGRAPHY, 4825
DIAGNOSIS, 4825
ORAL CONTRACEPTIVES, 4790*
REVIEWS, 4819
NEOPLASMS, MALIGNANT
ALCOHOLISM, 5094
ANTIGEN, AUSTRALIA, 4814, 4826
CATALASE, 4787*
CHEMOTHERAPY, 4781*, 4788*, 4829
ECHOGRAPHY, 4822
EPIDEMIOLOGY, 4794*
ETIOLOGY, 4820, 4826
FETOPROTEIN, ALPHA, 4014*, 4814, 4823
IMMUNOGLOBULINS, 4801
LAPAROSCOPY, 4269
NUCLEIC ACIDS, 4023
PATHOLOGY, 4820
PROGNOSIS, 4794*
REVIEWS, 4783*, 4819
SURGERY, 4788*, 4818, 4828
THERAPY, 4823
NUCLEIC ACIDS
CARBON DIOXIDE, 4022
ELECTROPHORESIS, 4173
SYNTHESIS, 4018, 4063
OBESITY
STARVATION, 4851
OBSTRUCTION
NEONATE, 4853
PANTOTHENIC ACID
METABOLISM, 4075
PATHOLOGY
CARBON TETRACHLORIDE, 4011*
CARCINOGENS, 4011*
FOOD ADDITIVES, 3917
SCLERODERMA, 4841
SHUNT, INTESTINAL, 4795*
THALASSEMIA, 4839
THYROID, 4850
PERFUSION
TECHNIQUES, 4100, 4102
PHOSPHORYLATION, OXIDATIVE
COENZYMES, 4095
PHENOBARBITAL, 4067
PORPHYRIA
THERAPY, 4845

LIVER (continued)
PORTACAVAL SHUNT
ANGIOGRAPHY, 4836
POTASSIUM
HORMONE CONTROL, 3995*
PROTEIN
MALNUTRITION, 4042
METABOLISM, 4089
SYNTHESIS, 4018, 4054, 4068, 4087, 4088
REGENERATION
AMINO ACIDS, 4017
CARBON TETRACHLORIDE, 4060
ENZYMES, 4021
EPINEPHRINE, 4146
FEEDING, 4021
HEPATECTOMY, 4015*, 4021
MITOCHONDRIA, 4017, 4147
NUCLEIC ACIDS, 4016, 4018
PROTEIN, 4016, 4018, 4019, 4142, 4147
SERUM, 4105
TRANSPORT, 4146
RUPTURE
ORAL CONTRACEPTIVES, 4790*
SCHISTOSOMIASIS
COMPLICATIONS, 5145
DIAGNOSIS, 5145
REVIEWS, 5144
SECRETION
MOTILITY, 3987
OBSTRUCTION, 4853
STEROID
METABOLISM, 4128
TRANSPLANTATION
IMMUNOSUPPRESSION, 4027
LIVER COMA, 4028, 4031
MAGNESIUM, 4074
MORPHOLOGY, 4030
PERFUSION, 4028
SURVIVAL, 4024
TECHNIQUES, 4024, 4025, 4026, 4029, 4074
TEMPERATURE, 4030
TRAUMA
COMPLICATIONS, 4835
SURGERY, 4832, 4834, 4835
TRIGLYCERIDE
DIABETES, 4844
FATTY LIVER, 4844
STARVATION, 4046
ULTRASTRUCTURE, 3789*
ADENYL CYCLASE, 3794*
CORTICOSTEROIDS, 4065
DEHYDRATION, 4053
DRUG EFFECTS ON, 4065
DRUG METABOLISM, 3787*
ENZYMES, 3797
HEPATECTOMY, 4143
HEPATITIS, CHRONIC, 4908
HEPATITIS, VIRAL, 4892
HORMONE, 3794*
IRRADIATION, 3790*
LEAD, 4064
STARVATION, 4851
UREA
SYNTHESIS, 4050, 4094
VITAMIN A
DEFICIENCY, 4090

LIVER (continued)
WILSONS DISEASE
CHILDREN, 4785*
ZINC
METABOLISM, 4069

LIVER COMA
DRUG-INDUCED
ANTIBACTERIALS, 4865
ANTIBIOTICS, 4865
DRUG TREATMENT, 4935
INFECTION, BACTERIA
CIRRHOSIS, 4912
LIVER
TRANSPLANTATION, 4028, 4031
PERFUSION, 4895
SIMULATION
ENZYMES, 4111
PATHOLOGY, 4111

LIVER DISEASE
ANGIOGRAPHY, 4270, 4810
ANTIBIOTICS
COMPLICATIONS, 4798
METABOLISM, 4798
ANTIGEN, AUSTRALIS
LIVER DISEASE, 4813
RADIOIMMUNOASSRY, 4813
BILE
PIGMENTS, 4103
SECRETION, 4806
BLEEDING
THERAPY, 4804
TRANSFUSION, 4804
VASOPRESSIN, 4804
BLOOD
ALCOHOL DEHYDROGENASE, 4812
CLOTTING, 4980
PLATELETS, 4803
CALCULI
SURGERY, 4847
CIRCULATION
COMPLICATIONS, 4852
COMPLICATIONS
NERVOUS SYSTEM, 4807
DIAGNOSIS, 4796*, 4809
ENZYMES, 4214
PHOSPHATASE, ALKALINE, 4263
DRUG METABOLISM
TRANQUILIZERS, 4793*
DRUG TREATMENT, 4800
COMPLICATIONS, 4805
ELECTROLYTE
CIRCULATION, 4103
FETOPROTEIN, ALPHA
EPIDEMIOLOGY, 4815
HEMODIALYSIS
SEQUELAE, 4791*
HEPATECTOMY, 4833
KIDNEY
CIRCULATION, 4103
NEOPLASMS, BENIGN, 4848
LAPAROSCOPY, 4808
LIVER
BILE DUCT, 4799
LIVER DISEASE
ANTIGEN, AUSTRALIA, 4813

LIVER DISEASE (continued)
LIVER FUNCTION TESTS
PATHOLOGY, 4292
REVIEWS, 4292
NEOPLASMS
SURGERY, 4847
NUCLEIC ACIDS
METABOLISM, 4023
PATHOLOGY
ANGIOGRAPHY, 4294
BIOPSY, 4294
PHOSPHATASE, ALKALINE
DIAGNOSIS, 4232
RADIOLOGY
REVIEWS, 4271
TECHNIQUES, 4213
SCANNING, SCINTILLATION, 4810
SCLERODERMA
AUTOIMMUNITY, 4904*
SMALL INTESTINE
SURGERY, 4780*
STENOSIS
SURGERY, 4847
STEROID
METABOLISM, 4849
SURGERY
CHILDREN, 4834

LIVER DISEASE, ALCOHOLIC
DIAGNOSIS
IMMUNOGLOBULINS, 4914
TRANSFERRIN, 4914
DISEASES ASSOCIATED WITH
CHOLESTASIS, 4916
FOLIC ACID
DEFICIENCY, 4913
LIPOPROTEINS
LIPASE, 4915
SECRETION, 4915
PATHOLOGY
OBESITY, 4780*

LIVER FUNCTION TESTS
ATHEROSCLEROSIS
AGE FACTORS, 4842
BLEEDING, 4145
CHOLESTASIS
PREGNANCY, 4955*
CYSTIC FIBROSIS, 4920*
GASTROENTERITIS
NEONATE, 5087
HYPERTENSION
AGE FACTORS, 4842
INDOCYANINE GREEN, 4260
LIVER
METASTASES, 4830
LIVER DISEASE
PATHOLOGY, 4292
REVIEWS, 4292
NEONATE, 4811
SCLERODERMA, 4841

LIVER INJURY
CARBON TETRACHLORIDE
BILIRUBIN, 4124
ISOENZYMES, 4061
LYSOSOMES, 4110
PREVENTION, 4059

LIVER INJURY (continued)
DRUG-INDUCED
ANESTHETICS, 4857*
ANTIBACTERIALS, 4867
EPIDEMIOLOGY
PREGNANCY, 4868
HYDROCARBONS, HALOGENATED
METABOLISM, 4062
LIPIDS
PEROXIDATION, 4076
PREVENTION
AMIDES, 4107
AMINO ACIDS, 4107

LYMPH
CIRCULATION
PORTAL HYPERTENSION, 4261

LYMPHANGIECTASIA
SMALL INTESTINE
CHILDREN, 4544*
TRIGLYCERIDE, MEDIUM CHAIN, 4543*

LYMPHATICS
BILE DUCT
COMPRESSION, 5008
COLON
POLYPS, 4680
GASTROINTESTINAL DISEASE, 5055
HODGKINS DISEASE
ENDOSCOPY, 4261
ILEUM
POLYPS, 4680
LIVER
ANGIOGRAPHY, 4817
STASIS, 4104
NEOPLASMS, MALIGNANT
ENDOSCOPY, 4261
GASTROINTESTINAL TRACT, 5048
PORTAL HYPERTENSION, 4039
SMALL INTESTINE
HYPERPLASIA, 4444
STOMACH
HYPERPLASIA, 4444

LYMPHOCYTE
HEPATITIS, CHRONIC
CORTICOSTEROIDS, 4871*
HEPATITIS, VIRAL
CORTICOSTEROIDS, 4871*
LYSOSOMES
HEPATITIS, CHRONIC
SIMULATION, 4110
LIVER
PHOSPHATASE, ACID, 4041
LIVER INJURY
CARBON TETRACHLORIDE, 4110

MACROGLOBULINEMIA
STOMACH
ENDOSCOPY, 4473

MAGNESIUM
ILEUM
SEROTONIN, 3875*
LIVER
TRANSPLANTATION, 4074
METABOLISM
INFLAMMATORY BOWEL DISEASE, 5191

MALABSORPTION
BILE ACIDS
METABOLISM, 4617
BLOOD
VITAMIN A, 4262
CROHNS DISEASE
VITAMIN D, 5195
DIAGNOSIS
AGE FACTORS, 4616
TOLERANCE TEST, 4190*
DISACCHARIDE
ANTINEOPLASTIC AGENTS, 4157
DRUG-INDUCED
ALCOHOL, 4641
DIETARY FACTORS, 4641
PATHOLOGY, 4641
REVIEWS, 4641
DUODENUM
ENTEROKINASE, 4612*
GLUTEN
REVIEWS, 4613
INTESTINE
ALCOHOLISM, 3835
JEJUNUM
ENTEROKINASE, 4612*
SMALL INTESTINE
DISACCHARIDASE, 5049
PATHOLOGY, 4620, 4622, 4638
THERAPY, 5047
AGE FACTORS, 4616
URINE
OXALATE, 5041
VITAMIN A
METABOLISM, 4618

MALIGNANT
SEE NEOPLASMS, MALIGNANT

MALNUTRITION
DRUG TREATMENT
COMPLICATIONS, 5023
LIVER
PROTEIN, 4042
THERAPY
CHILDREN, 5047

MANOMETRY
SEE PRESSURE STUDIES

MARASMUS
DISEASES ASSOCIATED WITH
RESPIRATORY SYSTEM, 5078

MECKELS DIVERTICULUM
ABDOMEN, ACUTE
ETIOLOGY, 4575
BLEEDING
ANGIOGRAPHY, 4545*
SCANNING, SCINTILLATION, 4545*
MORPHOLOGY
DIVERTICULUM, 4599
NEOPLASMS, BENIGN
BLEEDING, 4578
PEPTIC ULCER, 4578
PERFORATION
COMPLICATIONS, 4574
NEONATE, 4576
SCANNING, SCINTILLATION, 4186*

MEDIASTINUM
INFLAMMATION
ESOPHAGUS DISEASE, 4347
PSEUDOTUMOR
PORTAL HYPERTENSION, 4929

MEGACOLON
ANORECTUM
PRESSURE STUDIES, 4651*
CHILDREN
SURGERY, 4653*
DEFECATION
PRESSURE STUDIES, 4645*

MELENA
GASTROINTESTINAL
BLEEDING, 5126

MERCAPTANS
STOMACH
PEPTIC ULCER, 4512*

MERYCISM
DEGLUTITION
REVIEWS, 4309

METAPLASIA
INTESTINE
ENDOSCOPY, 4192*
SMALL INTESTINE
DISEASES ASSOCIATED WITH, 4360

METASTASES
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 4647*
LIVER, 4198*
ENZYMES, 4830
LIVER FUNCTION TESTS, 4830
SMALL INTESTINE
THERAPY, 4561
STOMACH
NEOPLASMS, MALIGNANT, 4461, 4465

MICROORGANISMS
ANTIBIOTICS
STIMULATION, 4183
CHOLECYSTITIS
EMPHYSEMA, 4943*
INTESTINE
MORPHOLOGY, 5157
LARGE INTESTINE
ULCERATIVE COLITIS, 4716
SMALL INTESTINE
ANTIBIOTICS, 4171

MICROSOMES
CARBOHYDRATE
METABOLISM, 4007*
DRUG METABOLISM
LIVER, 4052
LIVER
ENZYMES, 4084

MITOCHONDRIA
LIVER
REGENERATION, 4017, 4147
ULTRASTRUCTURE, 4054

MITOSIS
INTESTINE
CIRCADIAN RHYTHM, 4179
RADIATION, 4179
LIVER
CARCINOGENS, 4034
EPINEPHRINE, 4144
STRESS, 4144
SURGERY, 4141

MONOAMINOXIDASE
SMALL INTESTINE
ABSORPTION, 4158

MORTALITY
SEE SURVIVAL

MOTILITY
ANTRUM
VAGOTOMY, 3866*
CECUM
ADENOSINE TRIPHOSPHATE, 3907
COLON
BIOPOTENTIALS, 3904
BRADYKININ, 3864*
DRUG EFFECTS ON, 3903
ELECTROPHYSIOLOGY, 3910
NERVOUS CONTROL, 3864*
PROSTAGLANDIN, 3899, 3903
REVIEWS, 3908
DUODENUM
BIOPOTENTIALS, 3901
ELECTRICAL CONTROL, 3901
PANCREAS DISEASE, 3882
VAGOTOMY, 3883
ESOPHAGUS
CHOLECYSTOKININ, 3905
GALLBLADDER
ANOMALY, CONGENITAL, 4958
CALCIUM, 4736
CELIAC DISEASE, 4609*
CHOLELITHIASIS, 4944*
PANCREAS DISEASE, 3882
GASTROINTESTINAL
BIOPOTENTIALS, 3878
CHOLECYSTECTOMY, 3879
ELECTRICAL CONTROL, 3879
GASTRECTOMY, 3909
HORMONE EFFECTS ON, 3877
PANCREATITIS, 4165*
TECHNIQUES, 3880
VOMITING, 3878
ILEUM
ACETYLCHOLINE, 3870*
DRUG EFFECTS ON, 3890
ELECTRICAL CONTROL, 3870*
NARCOTICS, 3890, 3891
SEROTONIN, 3875*
INTESTINE
ANTIBIOTICS, 5013*
ELECTROPHYSIOLOGY, 3895
NERVOUS SYSTEM, 3892
TECHNIQUES, 3893
JEJUNUM
ANGIOTENSIN, 3884
LIVER
SECRETION, 3987

MOTILITY (continued)
PANCREAS
ATROPHY, 3882
SECRETION, 3987
SMALL INTESTINE
ANTISPASMODICS, 3896
ATROPINE, 3863*
CATECHOLAMINES, 3874*
CERULEIN, 3863*
ILEUS, 3906
NERVOUS CONTROL, 3900
NERVOUS SYSTEM, 3876*
PROSTAGLANDIN, 3881, 3899
SURGERY, 3902
VAGOTOMY, 3868*
STOMACH
ACIDS, 3889
ANALGESICS, 4377*
ANGIOTENSIN, 3884
BRADYKININ, 3864*
CATECHOLAMINES, 3871*, 3874*
GASTRIN, 3888
HORMONE EFFECTS ON, 3887
NERVOUS CONTROL, 3864*
NERVOUS SYSTEM, 3885
PANCREAS DISEASE, 3882
PEPTIC ULCER, 4506*
PREGNANCY, 4377*
PROSTAGLANDIN, 3881
PYLOROPLASTY, 4526
REVIEWS, 3886
SECRETIN, 3888
VAGOTOMY, 3883, 4526

MOUTH
NEOPLASMS, BENIGN
GENETIC FACTORS, 5021
SURGERY, 5021

MUCOPOLYSACCHARIDE
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 4685

MUSCULOSKELETAL SYSTEM
CROHN'S DISEASE
DISEASES ASSOCIATED WITH, 5170*

MYCOSIS
SEE INFECTION, FUNGUS

MYELOMA
STOMACH
DIAGNOSIS, 4290
RADIOLOGY, 4290

NARCOTICS
BILE
SECRETION, 4132
BILE ACIDS
SYNTHESIS, 4132
ILEUM
MOTILITY, 3890, 3891
PANCREAS
SECRETION, 3980*

NECROSIS
HEPATITIS, VIRAL
SURVIVAL, 4875*

NECROSIS (continued)
LIVER
BLEEDING, 4937
DIETARY FACTORS, 4071
FIBRINOLYSIS, 4886
GALACTOSAMINE, 4115
PREVENTION, 4071
TRACE ELEMENTS, 4071
PANCREATITIS
PANCREATECTOMY, 4747

NEONATE
SEE ALSO AGE FACTORS, CHILDREN
CARBOHYDRATE
ABSORPTION, 4608*
METABOLISM, 4608*
DIARRHEA, 5031
LACTATE DEHYDROGENASE, 5050
NUTRITION, 5018*
DISACCHARIDASE
INTOLERANCE, 5045
ENTEROCOLITIS, NECROTIZING
SURGERY, 5084
THERAPY, 5084
ESOPHAGUS
STENOSIS, 4336
GASTROENTERITIS
AMINO ACIDS, 5087
ESCHERICHIA COLI, 5112
LIVER FUNCTION TESTS, 5087
GASTROINTESTINAL
PERFORATION, 5104
HERNIA, HIATUS
RADIOLOGY, 4324
HIRSCHSPRUNGS DISEASE
RADIOLOGY, 4662
IRON
ABSORPTION, 3828
LIVER
OBSTRUCTION, 4853
LIVER FUNCTION TESTS, 4811
MECKELS DIVERTICULUM
PERFORATION, 4576
PYLORUS
STENOSIS, 4470
SMALL INTESTINE
KINETICS, CELL, 3801
OBSTRUCTION, 4548

NEOPLASMS
BILE DUCT
DRAINAGE, 4977
COLON
IMMUNITY, 4690
SURGERY, 4690
ENDOCRINE SYSTEM
GASTROINTESTINAL TRACT, 4283
PATHOLOGY, 5118
RADIOIMMUNOASSAY, 4283
RADIOLOGY, 4284
ESOPHAGUS
CARCINOIDS, 4350
DISEASES ASSOCIATED WITH, 4347
GASTROINTESTINAL
ENDOCRINE SYSTEM, 5114, 5116
IMMUNOLOGY, 5028
SEQUELAE, 4430
GASTROINTESTINAL TRACT
PATHOLOGY, 5118

NEOPLASMS (continued)
JAUNDICE, OBSTRUCTIVE
DIAGNOSIS, 5069
JEJUNUM
PERFORATION, 4559
KINETICS, CELL, 5095
LIVER
DIAGNOSIS, 4824
FETOPROTEIN, ALPHA, 4821
SCANNING, SCINTILLATION, 4821
SURGERY, 4827
LIVER DISEASE
SURGERY, 4847
PANCREAS
TECHNIQUES, 4199*
ZOLLINGER-ELLISON SYNDROME, 5116
SALIVARY GLANDS
CHILDREN, 5109, 5111
RADIOLOGY, 5109
SURGERY, 5111
SMALL INTESTINE
DIAGNOSIS, 4560
SURGERY, 4565
STOMACH
DIAGNOSIS, 4452
SURGERY, 4452

NEOPLASMS, BENIGN
SEE ALSO POLYPS
CECUM, 4707
COLON
ENDOSCOPY, 4238
DUODENUM, 4551
ESOPHAGUS
PATHOLOGY, 4344
GALLBLADDER
DIAGNOSIS, 4963
KIDNEY
LIVER DISEASE, 4848
LIVER
ANGIOGRAPHY, 4825
DIAGNOSIS, 4825
ORAL CONTRACEPTIVES, 4790*
REVIEWS, 4819
MECKELS DIVERTICULUM
BLEEDING, 4578
PEPTIC ULCER, 4578
MOUTH
GENETIC FACTORS, 5021
SURGERY, 5021
SMALL INTESTINE, 4563
ANEMIA, 4564
ENDOSCOPY, 4293
RUPTURE, 4562
STOMACH
ENDOSCOPY, 4293, 4474
FISTULA, 4431
RADIOLOGY, 4474

NEOPLASMS, MALIGNANT
ANORECTUM
COMPLICATIONS, 4697
RADIOLOGY, 4696
SURGERY, 4697
ANUS
CHEMOTHERAPY, 4692
RADIOTHERAPY, 4692
SURGERY, 4692

NEOPLASMS, MALIGNANT (continued)
APPENDIX, 4686
BILE DUCT
CHEMOTHERAPY, 4947*
RADIOTHERAPY, 4947*
SURGERY, 4947*, 5007
CELIAC DISEASE
COMPLICATIONS, 4636, 4643
SMALL INTESTINE, 4636
COLON
ANTIGEN, CARCINOEMBRYONIC, 4244, 4646*
CHEMOTHERAPY, 4657*
COMPLICATIONS, 4695
CROHNS DISEASE, 5193
DIAGNOSIS, 4699
DRUG TREATMENT, 4657*
ENDOSCOPY, 4237, 4238
EPIDEMIOLOGY, 4688, 4699
ETIOLOGY, 4699
IMMUNOLOGY, 4648*, 4687
INFECTION, 4694
ISCHEMIA, 4695
KIDNEY, 4698
PROGNOSIS, 4648*
RECURRENCE, 4687, 4689
SURGERY, 4649*
DUODENUM
ETIOLOGY, 4598
ENDOSCOPY
LYMPHATICS, 4261
ESOPHAGUS
ALCOHOLISM, 5094
CHEMOTHERAPY, 4348
DIAGNOSIS, 4351, 4354
DISEASES ASSOCIATED WITH, 4360, 4447
EMBRYOLOGY, 4345
PROGNOSIS, 4351
RADIOLOGY, 4348
RADIOTHERAPY, 4346, 4348, 4355
RECURRENCE, 4348
SURGERY, 4346, 4349, 4352, 4353
GALLBLADDER
DIARRHEA, 4994
FISTULA, 4994
PERFORATION, 4994
GASTROINTESTINAL
ALCOHOLISM, 5094
ASBESTOS, 4176
ENDOSCOPY, 4289
SURGERY, 5065
INTESTINE
ANTIGEN, CARCINOEMBRYONIC, 4178
LARGE INTESTINE
COMPLICATIONS, 4693
CROHNS DISEASE, 5180
DIAGNOSIS, 4647*
METASTASES, 4647*
MUCOPOLYSACCHARIDE, 4685
OBSTRUCTION, 4693
PERFORATION, 4693
PREVENTION, 4678
LIVER
ALCOHOLISM, 5094
ANTIGEN, AUSTRALIA, 4814, 4826
CATALASE, 4787*
CHEMOTHERAPY, 4781*, 4788*, 4829

NEOPLASMS, MALIGNANT (continued)
LIVER (continued)
ECHOGRAPHY, 4822
EPIDEMIOLOGY, 4794*
ETIOLOGY, 4820, 4826
FETOPROTEIN, ALPHA, 4014*, 4814, 4823
IMMUNOGLOBULINS, 4801
LAPAROSCOPY, 4269
NUCLEIC ACIDS, 4023
PATHOLOGY, 4820
PROGNOSIS, 4794*
REVIEWS, 4783*, 4819
SURGERY, 4788*, 4818, 4828
THERAPY, 4823
LYMPHATICS
GASTROINTESTINAL TRACT, 5048
PANCREAS
ALCOHOLISM, 5094
ANGIOGRAPHY, 4724
BIOPSY, 4188*
LIPASE, 4253
NEOPLASMS, MALIGNANT, 4253
PANCREATECTOMY, 4722*
SALIVARY GLANDS, 5063
PATHOLOGY, 5101
ULTRASTRUCTURE, 5101
SMALL INTESTINE, 4566
ANEMIA, 4601
CROHNS DISEASE, 5180
DIAGNOSIS, 4555
OBSTRUCTION, 4554
SURGERY, 4558, 4588
ULTRASTRUCTURE, 4554
STOMACH, 4457
AGE FACTORS, 4393, 4453, 4460
ALCOHOLISM, 5094*
BIOPSY, 4449
BLEEDING, 4461
CIRRHOSIS, 4927
CYTOLOGY, 4224, 4389*
DIAGNOSIS, 4445, 4451, 4454, 4464
DISEASES ASSOCIATED WITH, 4447
ENDOSCOPY, 4192*, 4196*, 4224, 4383*, 4448, 4449
ENVIRONMENTAL FACTORS, 4442
EPIDEMIOLOGY, 4460, 4467
ETIOLOGY, 4463
FIBRINOLYSIS, 4394
GASTRECTOMY, 4456
GLYCOPROTEINS, 4443
HETEROTOPIA, 4574
KINETICS, CELL, 4412
LACTATE DEHYDROGENASE, 4428
METASTASES, 4461, 4465
MORPHOLOGY, 4432, 4463
PATHOLOGY, 4368*, 4446, 4465
PHOSPHATASE, ACID, 4367*
PRECANCER, 4458, 4459, 4466
PROGNOSIS, 4454, 4464
RADIOLOGY, 4223
RADIOTHERAPY, 4441
RECURRENCE, 4456
REVIEWS, 4468
SEX FACTORS, 4453, 4460
STATISTICAL STUDY, 4467
SURGERY, 4440, 4445, 4462
SURVIVAL, 4451
THERAPY, 4454, 4464

NERVOUS CONTROL
ANTRUM
GASTRIN, 3936*
COLON
MOTILITY, 3864*
DEGLUTITION, 4310
ESOPHAGUS
PERISTALSIS, 3867*
PANCREAS
SECRETION, 3977*
PERISTALSIS
ILEUM, 3897
SMALL INTESTINE
MOTILITY, 3900
STOMACH
ACID SECRETION, 4396
MOTILITY, 3864*

NERVOUS SYSTEM
BILE DUCT
MORPHOLOGY, 4127
CIRRHOSIS
DRUG TREATMENT, 4936
ESOPHAGUS
CHEMORECEPTION, 3919
MORPHOLOGY, 3919
GALLBLADDER
CHOLECYSTITIS, 4135
GASTROINTESTINAL TRACT, 4175
HIRSCHSPRUNGS DISEASE
COMPLICATIONS, 4662
INTESTINE
ABSORPTION, 3827
MOTILITY, 3892
LIVER DISEASE
COMPLICATIONS, 4807
SMALL INTESTINE
MOTILITY, 3876*
OBSTRUCTION, 4596
STOMACH
MOTILITY, 3885

NITROGEN
DIGESTION
LACTOSE, 4153*

NITROSAMINES
METABOLISM, 3818*

NUCLEIC ACIDS
BILIARY TRACT
CORTICOSTEROIDS, 4131
LIVER
CARBON DIOXIDE, 4022
ELECTROPHORESIS, 4173
NEOPLASMS, MALIGNANT, 4023
REGENERATION, 4016, 4018
METABOLISM
CARBON DIOXIDE, 4022
INTESTINE, 4163
LIVER DISEASE, 4023
VITAMIN D, 4163
PROTEIN
METABOLISM, 4089
STOMACH
ELECTROPHORESIS, 4173
SYNTHESIS
CARCINOGENS, 3993, 4081

NUCLEIC ACIDS (continued)
SYNTHESIS (continued)
CORTICOSTEROIDS, 4131
FUNGICIDES, 4063
HEPATECTOMY, 4015*, 4020
LIVER, 4018, 4063
SALIVARY GLANDS, 3912
SEX FACTORS, 4081

NUCLEOTIDES
GLUCONEOGENESIS
LIVER, 4080

NUTRITION
DIARRHEA
NEONATE, 5018*
LARGE INTESTINE
FISTULA, 4672
SMALL INTESTINE
SURGERY, 4177
OBESITY
CALCIUM
ABSORPTION, 4619
HERNIA, HIATUS, 4323
LIVER DISEASE, ALCOHOLIC
PATHOLOGY, 4780*
SMALL INTESTINE
SURGERY, 4593, 4594, 4780*
STARVATION
LIVER, 4851
SURGERY
FATTY LIVER, 4795*
SEQUELAE, 4780*
SIMULATION, 4160
TECHNIQUES, 4160

OBSTRUCTION
BILE DUCT
AMYLASE, 4197*
BIOPSY, 4948*
DIAGNOSIS, 4197*
ECHINOCOCCOSIS, 5139
SURGERY, 4990
UROGENITAL SYSTEM, 4006*
INTESTINE
GALLSTONE, 4570, 4571
SURGERY, 4571
LARGE INTESTINE
ANTICOAGULANTS, 4669
DISEASES ASSOCIATED WITH, 4686
DRUG-INDUCED, 4669
DRUG TREATMENT, 4668
NEOPLASMS, MALIGNANT, 4693
LIVER
NEONATE, 4853
SECRETION, 4853
PYLORUS
ANTIEMETICS, 4372*
SURGERY, 4573
SMALL INTESTINE
CROHNS DISEASE, 5193
HEMATOMA, 4569
NEONATE, 4548
NEOPLASMS, MALIGNANT, 4554
NERVOUS SYSTEM, 4596
SURGERY, 4573
STOMACH
ANOMALY, 4477
PYLORUS, 4477

OBSTRUCTIVE JAUNDICE
SEE JAUNDICE, OBSTRUCTIVE

OCCUPATIONAL FACTORS
CADMIUM
ANALYSIS, 4866
LIVER, 4866

ODDI, SPHINCTER OF
BIOPOTENTIALS
DRUG EFFECTS ON, 3865*
DISEASES
PRESSURE STUDIES, 4295
DRUG EFFECTS ON, 4973
SURGERY
RECURRENCE, 4959

OMENTUM
TORSION
SURGERY, 5064

ORAL CONTRACEPTIVES
CHOLELITHIASIS
EPIDEMIOLOGY, 4951*
LIVER
NEOPLASMS, BENIGN, 4790*
RUPTURE, 4790*

OXALATE
ABSORPTION
GASTROINTESTINAL DISEASE, 5026
URINE
CIRRHOSIS, 5041
GASTROINTESTINAL DISEASE, 5041
MALABSORPTION, 5041

OXIDATION
ALCOHOL
CATALASE, 3997*
LIVER, 3996*, 3997*, 4049
FATTY ACIDS
ALCOHOL, 4154*
SMALL INTESTINE, 4154*
PENTOSE
ACID SECRETION, 3950
STOMACH, 3950

OXYGEN
STOMACH
PERMEABILITY, 3844

PANCREAS
ANOMALY, CONGENITAL
SURGERY, 4725
ATROPHY
MOTILITY, 3882
BIOPSY
PANCREATITIS, 4774
TECHNIQUES, 4199*
CIRCULATION
ELECTRICAL CONTROL, 4166*
PANCREATITIS, CHRONIC, 4745*
CYSTS
COMPLICATIONS, 4729
ETIOLOGY, 4770
RADIOLOGY, 4743*
SURGERY, 4770

PANCREAS (continued)
ENZYMES
ANTIBACTERIALS, 3991
ANTIBIOTICS, 3991
CALCIUM, 4736
CHEMICAL COMPOSITION, 3974*
SECRETION, 4736
STARVATION, 3989
GROWTH
HORMONE, 3785*
HETEROTOPIA
DUODENUM, 4733
WHIPPLES DISEASE, 4625
METABOLISM
ALCOHOL, 3982
MORPHOLOGY
PANCREATITIS, 3796
NEOPLASMS
TECHNIQUES, 4199*
ZOLLINGER-ELLISON SYNDROME, 5116
NEOPLASMS, MALIGNANT, 4253
ALCOHOLISM, 5094
ANGIOGRAPHY, 4724
BIOPSY, 4188*
LIPASE, 4253
PANCREATECTOMY, 4722*
PATHOLOGY
CARCINOGENS, 3993
PSEUDOCYST
COMPLICATIONS, 4726
DRAINAGE, 4728, 4776
ECHOGRAPHY, 4727
REVIEWS, 4769
SURGERY, 4726, 4762, 4768, 4771
RADIOLOGY, 4253
SCANNING, SCINTILLATION
TECHNIQUES, 4249
SECRETION
AGE FACTORS, 4773
ALCOHOL, 3982, 4767
ALCOHOLISM, 4766
AMINES, 3979*
ANTIBIOTICS, 3983
BILE, 3985
CALCIUM, 3986
CHOLECYSTOKININ, 3986
DRUG EFFECTS ON, 3983
H2 RECEPTOR ANTAGONISTS, 4483*
HORMONE CONTROL, 3952
HYPOXIA, 3978*
MOTILITY, 3987
NARCOTICS, 3980*
NERVOUS CONTROL, 3977*
PANCREATITIS, 4766
PANCREOZYMIN, 3986
PEPTIC ULCER, 4483*
PROTEIN, 3976*, 3990
SYMPATHOMIMETIC AGENTS, 3979*
TRANSPLANTATION, 3984
VAGOTOMY, 3981*
SMALL INTESTINE
SURGERY, 4177
SODIUM
METABOLISM, 3809*
SURGERY
COMPLICATIONS, 4731
SEQUELAE, 5059
TECHNIQUES, 4731

PANCREAS (continued)
TRANSPLANTATION
PANCREATITIS, 4740*
TRAUMA
ENZYMES, 3992
SURGERY, 4732

PANCREAS DISEASE
ALCOHOL
REVIEWS, 4767
ALCOHOLISM, 4723*
REVIEWS, 4730
ARTERY
EROSION, 4729
DIAGNOSIS
CHILDREN, 4245
ENZYMES, 4214
DUODENUM
MOTILITY, 3882
ENDOSCOPY, 4252, 4255, 4256, 4257
COMPLICATIONS, 4258
SURGERY, 4254
FECES
CHYMOTRYPSIN, 4246, 4247
GALLBLADDER
MOTILITY, 3882
GASTROINTESTINAL
BLEEDING, 4729
RADIOLOGY, 4233, 4250, 4251
TECHNIQUES, 4213
SCANNING, SCINTILLATION, 4250
STOMACH
MOTILITY, 3882
SURGERY
BIOPSY, 4188*
DISEASES ASSOCIATED WITH, 4775
VITAMIN A
METABOLISM, 4618
ZINC
SECRETION, 4735

PANCREAS FUNCTION TESTS
CYSTIC FIBROSIS, 4734
DRUG EFFECTS ON
ANTIBIOTICS, 4772
PANCREATITIS, CHRONIC
SURGERY, 4762
SMALL INTESTINE
SURGERY, 4177

PANCREATECTOMY
IRON
ABSORPTION, 5059
PANCREAS
NEOPLASMS, MALIGNANT, 4722*
PANCREATITIS
NECROSIS, 4747

PANCREATIC DUCT
PATHOLOGY
REVIEWS, 5068
SURGERY
REVIEWS, 5068

PANCREATITIS
ALCOHOL
THERAPY, 4764

PANCREATITIS (continued)
ASCITES
PHOSPHOLIPASE, 4741*
BICARBONATE
SECRETION, 4754
BILE DUCT
STRICTURE, 4988
BLEEDING
DRUG TREATMENT, 4750
SURGERY, 4750, 4752
BLOOD
CLOTTING, 4753
FATTY ACIDS, 4755
HISTAMINE, 4756
LIPIDS, 4755
PHOSPHOLIPASE, 4741*
SEROTONIN, 4756
COLON
STENOSIS, 4664
COMPLICATIONS
REVIEW, 4777
CROHNS DISEASE
DISEASES ASSOCIATED WITH, 5193
DIAGNOSIS
AMYLASE, 4197*
COMPLICATIONS, 4746
REVIEW, 4777
DISEASES ASSOCIATED WITH
CHOLELITHIASIS, 4744*
DRUG-INDUCED
ANTINEOPLASTIC AGENTS, 4758
DRUG TREATMENT
ANTIBIOTICS, 4739*
ANTIHISTAMINES, 4756
DUODENUM
BIOPSY, 5051
CIRCULATION, 4165*
ENDOSCOPY, 4252, 4255
EPIDEMIOLOGY
AGE FACTORS, 4778
RECURRENCE, 4778
SEX FACTORS, 4778
ETIOLOGY
ALCOHOLISM, 4777
FECES
CHYMOTRYPSIN, 4246
GASTROINTESTINAL
MOTILITY, 4165*
HYPERLIPEMIA
COMPLICATIONS, 4757
KIDNEY
TRANSPLANTATION, 4779
NECROSIS
PANCREATECTOMY, 4747
PANCREAS
BIOPSY, 4774
MORPHOLOGY, 3796
SECRETION, 4766
TRANSPLANTATION, 4740*
PREVENTION
ANTINEOPLASTIC AGENTS, 4740*
SALIVARY GLANDS
MORPHOLOGY, 3796
SIMULATION
ENZYMES, 3992
SURGERY
REVIEW, 4777
TECHNIQUES, 4748, 4749, 4751
THERAPY, 4746, 4761

PANCREATITIS, CHRONIC
BLOOD
LIPASE, 4253
CALCIFICATION
ALCOHOL, 4765
EPIDEMIOLOGY, 4742*
ETIOLOGY, 4742*
PATHOLOGY, 4737*
ENDOSCOPY
SURGERY, 4254
PANCREAS
CIRCULATION, 4745*
RADIOLOGY, 4760
RECURRENCE
CIRCULATION, 4763
SMALL INTESTINE
ELECTROLYTE, 5053
SURGERY
PANCREAS FUNCTION TESTS, 4762
REGENERATION, 4762
SEQUELAE, 4759
TECHNIQUES, 4738*, 4760

PANCREOZYMIN
PANCREAS
SECRETION, 3986

PANTOTHENIC ACID
METABOLISM
COENZYMES, 4075
LIVER, 4075

PAPILLA OF VATER
ENDOSCOPY, 4274
SCANNING, SCINTILLATION
TECHNIQUES, 4248

PARASITES AND PARASITIC DISEASE
ANTIGEN
ELECTROPHORESIS, 4193*
CHOLANGITIS
SURGERY, 5011
CHOLECYSTITIS
SURGERY, 5011
DIAGNOSIS
ELECTROPHORESIS, 4193*
DRUG TREATMENT, 5143
IMMUNOLOGY
DIAGNOSIS, 4282
KINETICS, 4170
REVIEWS, 5133
SOCIOECONOMIC FACTORS, 5153

PARATHYROID GLAND
STOMACH
SECRETION, 4378*

PARATYPHOID FEVER
SEE SALMONELLOSIS

PENTAGASTRIN
AMINO ACIDS
METABOLISM, 3933*
COMPLICATIONS
CIRCULATION, 3955
GALLBLADDER
PRESSURE STUDIES, 3869*
SMALL INTESTINE
KINETICS, CELL, 4155*

PENTAGASTRIN (continued)
STOMACH
PEPTIC ULCER, 4505*
SECRETION, 3955, 3957

PENTOSE
OXIDATION
ACID SECRETION, 3950
STOMACH, 3950

PEPSIN
SECRETION
BILE ACIDS, 3935*
HORMONE CONTROL, 3944*, 3951, 3954
HORMONE EFFECTS ON, 3887
STOMACH
ATROPINE, 4499*
BILE ACIDS, 3935*
ISOLATION, 3959
PEPTIC ULCER, 4505*, 4511*

PEPTIC ULCER
ACID SECRETION
ANTICHOLINERGIC AGENTS, 4491*
CALCIUM, 4507*
ETHNIC FACTORS, 4504*
H2 RECEPTOR ANTAGONISTS, 4491*
HISTAMINE, 4504*, 4507*
AGE FACTORS, 4485*
ANTACIDS, 4520
ANTICHOLINERGIC AGENTS, 4520
ANTRECTOMY, 4508*
BILE
STRESS, 4539
BILE ACIDS
STRESS, 4539
BLOOD
GASTRIN, 4488*
SECRETIN, 4490*
VITAMIN B6, 4482*
COMPLICATIONS
BLEEDING, 4535
DIAGNOSIS, 4516
DISEASES ASSOCIATED WITH, 4485*
RESPIRATORY SYSTEM, 4533
DRUG EFFECTS ON, 4521
DRUG-INDUCED
AGE FACTORS, 4538
ANTIINFLAMMATORY AGENTS, 4500*, 4538
HEALING, 4500*
VITAMIN C, 4500*
DRUG TREATMENT, 4518
CARBENOXALONE, 4370*
DRUG TOXICITY, 4182
ENDOSCOPY, 4227
EPIDEMIOLOGY, 4485*
ETIOLOGY, 4514, 4515
GASTRIN
ANTICHOLINERGIC AGENTS, 4491*
H2 RECEPTOR ANTAGONISTS, 4491*
SECRETION, 4471
GENETIC FACTORS, 4493*
HEALING, 4516
HISTAMINE
METABOLISM, 4502*
INTEGUMENTARY SYSTEM
TEMPERATURE, 4537

PEPTIC ULCER (continued)
JEJUNUM
VAGOTOMY, 4522
KIDNEY
TRANSPLANTATION, 4498*
MECKELS DIVERTICULUM
NEOPLASMS, BENIGN, 4578
PANCREAS
SECRETION, 4483*
PATHOLOGY, 4515
PERFORATION
RADIOLOGY, 4532
PREVENTION
DRUG TREATMENT, 4496*
PYLOROPLASTY, 4510*
RADIOLOGY, 4228
RECURRENCE, 4487*
ACHLORHYDRIA, 4509*
SURGERY, 4517
REVIEWS, 4513
SIMULATION
AMINES, 4501*
BILE ACIDS, 4539
DRUG TREATMENT, 4501*
SMALL INTESTINE
ELECTROLYTE, 5053
STOMACH
ACID SECRETION, 4488*, 4492*, 4503*, 4505*, 4511*, 4519
BLEEDING, 4421
ENDOSCOPY, 4415
LIPASE, 4505*
MERCAPTANS, 4512*
MORPHOLOGY, 4432
MOTILITY, 4506*
PATHOLOGY, 4489*, 4512*
PENTAGASTRIN, 4505*
PEPSIN, 4505*, 4511*
SECRETION, 4483*, 4494*, 4495*, 4499*, 4534
SURGERY
RADIOLOGY, 4527
REFLUX, 4484*
REVIEWS, 4528
SEQUELAE, 4484*, 4526, 4531
TECHNIQUES, 4497*
THERAPY, 4514
URINE
CATECHOLAMINES, 4536
STEROID, 4536
VAGOTOMY, 4508*, 4510*, 4521
ANTRECTOMY, 4525
COMPLICATIONS, 4592
ENTERITIS, 4592
PYLOROPLASTY, 4527
SEQUELAE, 4529
SURVIVAL, 4529
TECHNIQUES, 4523, 4524

PERFORATION
COLITIS
ISCHEMIA, 4703
COLON
ENDOSCOPY, 4666
ESOPHAGUS
CONTRAST MEDIA, 4195*
RADIOLOGY, 4340
SURGERY, 4341

PERFORATION (continued)
GALLBLADDER
NEOPLASMS, MALIGNANT, 4994
GASTROINTESTINAL
ETIOLOGY, 5104
NEONATE, 5104
HODGKINS DISEASE
DUODENUM, 4557
JEJUNUM
BIOPSY, 4589
NEOPLASMS, 4559
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 4693
MECKELS DIVERTICULUM
COMPLICATIONS, 4574
NEONATE, 4576
PEPTIC ULCER
RADIOLOGY, 4532
SMALL INTESTINE
SALMONELLOSIS, 5086
SURGERY, 4542*
ULCERATIVE COLITIS
PERITONITIS, 5071

PERFUSION
BILE DUCT
PRESSURE STUDIES, 4973
HEPATITIS, VIRAL
ATROPHY, 4895
LIVER
ENZYMES, 4140
TECHNIQUES, 4100, 4102
TRANSPLANTATION, 4028
LIVER COMA, 4895

PERISTALSIS
ESOPHAGUS
NERVOUS CONTROL, 3867*
ILEUM
NERVOUS CONTROL, 3897

PERITONEOSCOPY
ABDOMEN, ACUTE, 4189*

PERITONITIS
BILE, 5042
ULCERATIVE COLITIS
PERFORATION, 5071

PERMEABILITY
BILE DUCT
CYSTS, 4973
ESOPHAGUS
ACIDS, 4301*
BILE ACIDS, 4301*
ESOPHAGITIS, 4301*
INTESTINE
LAXATIVES, 3833
STOMACH
ACIDITY, 3844
ALCOHOL, 3938*
ANTIINFLAMMATORY AGENTS, 3938*
BILE ACIDS, 3938*
CIRCULATION, 3844
OXYGEN, 3844

PEROXIDATION
ALCOHOL
LIVER, 4002*
LIPIDS
LIVER, 3999*, 4052
LIVER INJURY, 4076

PERSORPTION
GASTROINTESTINAL TRACT, 3813*

PESTICIDES
PROTEIN
SYNTHESIS, 4068

PEUTZ-JEGHERS SYNDROME
RADIOLOGY, 4553, 4683
SURGERY, 4553, 4683

PHENOBARBITAL
BILE
SYNTHESIS, 3994*
BILIRUBIN
METABOLISM, 4124
CHOLESTASIS
DRUG TREATMENT, 4942*
CRIGLER-NAJJAR SYNDROME, 4856
LIPIDS
SECRETION, 3994*
LIVER
CARBON TETRACHLORIDE, 4011*
ENZYMES, 4067
ION TRANSPORT, 4148
PHOSPHORYLATION, OXIDATIVE, 4067

PHENYLKETONURIA
DISEASES ASSOCIATED WITH
DUODENITIS, 5103

PHOSPHATASE, ACID
LYSOSOMES
LIVER, 4041
STOMACH
NEOPLASM, MALIGNANT, 4367*

PHOSPHATASE, ALKALINE
BLOOD
BILE, 4004*
DEFICIENCY
INFECTION, 5098
SMALL INTESTINE, 5098
LIVER DISEASE
DIAGNOSIS, 4232, 4263
STOMACH
PRECANCER, 4387*

PHOSPHODIESTERASE
STOMACH
FEEDING, 3970
IRRADIATION, 3970

PHOSPHOLIPASE
ASCITES
CIRRHOSIS, 4741*
PANCREATITIS, 4741*
BLOOD
PANCREATITIS, 4741*

PHOSPHOLIPID
BILIRUBIN
METABOLISM, 4119
TRANSPORT, 4119
BLOOD
CIRRHOSIS, 4923*
HEPATITIS, CHRONIC, 4923*

PHOSPHOLIPIDS
CHOLELITHIASIS, 5003

PHOSPHORUS
FLUORIDE
METABOLISM, 3855

PHOSPHORYLATION, OXIDATIVE
LIVER
COENZYMES, 4095
PHENOBARBITAL, 4067

PHOTOTHERAPY
ASPIRIN
METABOLISM, 4854*
BILIRUBIN
METABOLISM, 4854*
CRIGLER-NAJJAR SYNDROME, 4856
HYPERBILIRUBINEMIA, 4855

PHYSICAL FACTORS
ESOPHAGUS
ACIDITY, 3862*
PRESSURE STUDIES, 3862*
STOMACH
ACIDITY, 3862*
PRESSURE STUDIES, 3862*

PIGMENTS
BILE
LIVER DISEASE, 4103

PLASMA
SEE BLOOD

PLATELETS
BLOOD
LIVER DISEASE, 4803

PNEUMATOSIS
LARGE INTESTINE, 4706

POLYPS
SEE ALSO NEOPLASMS, BENIGN
APPENDIX, 4691
COLON
ANGIOGRAPHY, 4677
ENDOSCOPY, 4676
LYMPHATICS, 4680
DUODENUM
RADIOLOGY, 4552
ESOPHAGUS
HAMARTOMA, 4343
FAMILIAL FACTORS
COLON, 4678
RECTUM, 4678
GASTROINTESTINAL, 5066
PROTEIN-LOSING ENTEROPATHY, 5043
ILEUM
LYMPHATICS, 4680

POLYPS (continued)
LARGE INTESTINE
SURGERY, 4721
STOMACH
ENDOSCOPY, 4222, 4371*

PORPHYRIA
LIVER
THERAPY, 4845

PORTACAVAL SHUNT
BILE
CHEMICAL COMPOSITION, 4136
EXCRETION, 4136
CIRRHOSIS
IMMUNITY, 4924*
IMMUNOLOGY, 4924*
INTESTINE
MORPHOLOGY, 4038
LIVER
ANGIOGRAPHY, 4836
CIRCULATION, 4040, 4933
MORPHOLOGY, 4038
ULCER
STRESS, 4439

PORTAL HYPERTENSION
BILIARY TRACT
SURGERY, 4957
DISEASES ASSOCIATED WITH
BILIARY DISEASE, 4957
LYMPH
CIRCULATION, 4261
LYMPHATICS, 4039
PSEUDOTUMOR
MEDIASTINUM, 4929
SURGERY
CHILDREN, 4939
VASOPRESSIN, 4918*

POTASSIUM
LIVER
HORMONE CONTROL, 3995*

PRECANCER
COLITIS
CROHNS DISEASE, 5196
STOMACH
DISACCHARIDASE, 4387*
GASTRITIS, 4422
NEOPLASMS, MALIGNANT, 4458, 4459, 4466
PATHOLOGY, 4446
PHOSPHATASE, ALKALINE, 4387*

PREGNANCY
CHOLESTASIS
LIVER FUNCTION TESTS, 4955*
LIVER
ABSCESS, 5137
LIVER INJURY
EPIDEMIOLOGY, 4868
STOMACH
MOTILITY, 4377*

PREMATURITY
CARBOHYDRATE
ABSORPTION, 4608*
METABOLISM, 4608*

PRIMATES
ASBESTOS
FEEDING, 4176

PROGNOSIS
COLON
NEOPLASMS, MALIGNANT, 4648*
ESOPHAGUS
NEOPLASMS, MALIGNANT, 4351
LIVER
NEOPLASMS, MALIGNANT, 4794*
STOMACH
NEOPLASMS, MALIGNANT, 4454, 4464

PROLAPSE
ESOPHAGUS, 4312

PROSTAGLANDIN
BLOOD
GASTRIN, 3967
COLON
MOTILITY, 3899, 3903
GALLBLADDER
PRESSURE STUDIES, 3869*
ISOLATION
GASTROINTESTINAL TRACT, 3916
SMALL INTESTINE
MOTILITY, 3881, 3899
STOMACH
ACID SECRETION, 3927*
MOTILITY, 3881

PROTEIN
ABSORPTION
AGE FACTORS, 3832
SMALL INTESTINE, 3832
BILIRUBIN
BINDING, 4181
BINDING
BILIRUBIN, 4118
CALCIUM, 4163
VITAMIN D, 4163
BLOOD
HEPATECTOMY, 4016
CELIAC DISEASE
GLUTEN, 4629
CHOLESTEROL
SYNTHESIS, 4043
DEFICIENCY
DISACCHARIDASE, 5090
DIGESTION
LACTOSE, 4153*
FECES
DIETARY FACTORS, 5092
HYDROLYSIS
GASTROINTESTINAL DISEASE, 5027
STOMACH DISEASE, 5027
IMMUNITY
CELIAC DISEASE, 4607*
SMALL INTESTINE, 4607*
INTOLERANCE
IMMUNOLOGY, 4621
SMALL INTESTINE, 4606*
LIVER
GROWTH FACTOR, 4019
MALNUTRITION, 4042
REGENERATION, 4016, 4018, 4019, 4142, 4147

PROTEIN (continued)
METABOLISM
CIRCULATION, 4852
HORMONE EFFECTS ON, 4089
LIVER, 4089
NUCLEIC ACIDS, 4089
PANCREAS
SECRETION, 3976*, 3990
SALIVARY GLANDS
FEEDING, 3914
SMALL INTESTINE
DISACCHARIDASE, 5090
STOMACH
ULCERATIVE COLITIS, 4714
SYNTHESIS
AGE FACTORS, 5096
CARCINOGENS, 4081
CORTICOSTEROIDS, 4065
DIETARY FACTORS, 5096
DRUG EFFECTS ON, 5023
FUNGICIDES, 4063
HORMONE EFFECTS ON, 4087
LIVER, 4018, 4054, 4068, 4087, 4088
PESTICIDES, 4068
SEX FACTORS, 4081
STARVATION, 4088
VITAMIN D, 4112
VITAMIN D
DEFICIENCY, 4112

PROTEIN-LOSING ENTEROPATHY, 4614
GASTROINTESTINAL
POLYPS, 5043

PROTEINS
HYPERLIPOPROTEINEMIA
LIVER, 4797*

PROTOZOA
INFECTION
EPIDEMIOLOGY, 5135
INTESTINE
EPIDEMIOLOGY, 5140

PSEUDOCYST
PANCREAS
COMPLICATIONS, 4726
DRAINAGE, 4728, 4776
ECHOGRAPHY, 4727
REVIEWS, 4769
SURGERY, 4726, 4762, 4768, 4771

PSEUDOTUMOR
ESOPHAGUS
INFLAMMATION, 4364
VARICES, 4359
MEDIASTINUM
PORTAL HYPERTENSION, 4929
SMALL INTESTINE
PATHOLOGY, 4541*
STOMACH
CHOLESTEROL, 4455

PSYCHOLOGICAL FACTORS
GASTROINTESTINAL DISEASE, 5056, 5057
TRANQUILIZERS, 5077

PYLOROPLASTY
PEPTIC ULCER, 4510*
VAGOTOMY, 4527
STOMACH
MOTILITY, 4526

PYLORUS
SEE ALSO STOMACH
OBSTRUCTION
ANTIEMETICS, 4372*
SURGERY, 4573
STENOSIS
ANOMALY, CONGENITAL, 4469
DIAGNOSIS, 4470
DISEASES ASSOCIATED WITH, 4547
NEONATE, 4470
THERAPY, 4470
STOMACH
ANOMALY, CONGENITAL, 4480
OBSTRUCTION, 4477

RADIATION
INTESTINE
MITOSIS, 4179

RADIOIMMUNOASSAY
ENDOCRINE SYSTEM
NEOPLASMS, 4283
HEPATITIS, VIRAL
ANTIGEN, 4878
ANTIGEN, AUSTRALIA, 4896
LIVER DISEASE
ANTIGEN, AUSTRALIA, 4813

RADIOISOTOPES
ESOPHAGUS DISEASE
SCANNING, SCINTILLATION, 4200*
HEPATITIS, VIRAL
VITAMIN B12, 4883
SMALL INTESTINE
ABSORPTION, 3838

RADIOLOGY
ANORECTUM
NEOPLASMS, MALIGNANT, 4696
BILE DUCT, 4277
CHOLECYSTITIS, 4940*
BILIARY
TECHNIQUES, 4273
BILIARY DISEASE, 4250
CELIAC DISEASE
BARIUM, 4630
CHILDREN, 4632
CHOLECYSTITIS
SURGERY, 4988
COLON
CONTRAST MEDIA, 4240
TECHNIQUES, 4243
CROHNS DISEASE, 5172*
DUODENUM
ANALGESICS, 4411
DISEASES, 4233
POLYPS, 4552
TECHNIQUES, 4234
DYSPHAGIA, 4300*

RADIOLOGY (continued)
ENDOCRINE SYSTEM
CARCINOIDS, 4284
NEOPLASMS, 4284
ESOPHAGITIS, REFLUX, 4315
ESOPHAGUS
DISEASES, 4315
DYSKINESIA, 4317
NEOPLASMS, MALIGNANT, 4348
PERFORATION, 4340
REFLUX, 4363
SCLERODERMA, 4338
STENOSIS, 4218
GALLBLADDER
BILIARY DISEASE, 4992
HETEROTOPIA, 4968
TECHNIQUES, 4280
GALLBLADDER DISEASE
CHOLECYSTOKININ, 4286
HYPERPLASIA, 4275
TECHNIQUES, 4213
GARDNERS SYNDROME, 4682
GASTROINTESTINAL, 4202
BLEEDING, 4207
HERNIA, HIATUS, 4315
CHILDREN, 4324
NEONATE, 4324
HIRSCHSPRUNGS DISEASE
NEONATE, 4662
LARGE INTESTINE
BLEEDING, 4212
LIVER DISEASE
REVIEWS, 4271
TECHNIQUES, 4213
PANCREAS, 4253
CYSTS, 4743*
PANCREAS DISEASE, 4233, 4250, 4251
TECHNIQUES, 4213
PANCREATITIS CHRONIC, 4760
PEPTIC ULCER, 4228
PERFORATION, 4532
SURGERY, 4527
PEUTZ-JEGHERS SYNDROME, 4553, 4683
RECTUM
BLEEDING, 4212
REGIONAL ENTERITIS, 5175
SALIVARY GLANDS
NEOPLASMS, 5109
SMALL INTESTINE
BLEEDING, 4212
INTUSSUSCEPTION, 4586
STOMACH
ANALGESICS, 4411
ATROPHY, 4373*
CONTRAST MEDIA, 4220
CROHNS DISEASE, 5168*
MYELOMA, 4290
NEOPLASMS, BENIGN, 4474
NEOPLASMS, MALIGNANT, 4223
TECHNIQUES, 4204, 4225
ULCERATIVE COLITIS
CIRCULATION, 3805

RADIOPROTECTIVE AGENTS
COPPER
METABOLISM, 4082

RADIOTHERAPY
ANUS
NEOPLASMS, MALIGNANT, 4692

RADIOTHERAPY (continued)
BILE DUCT
NEOPLASMS, MALIGNANT, 4947*
ESOPHAGUS
NEOPLASMS, MALIGNANT, 4346, 4348, 4355
STOMACH
NEOPLASMS, MALIGNANT, 4441

RECTUM
SEE ALSO LARGE INTESTINE
BIOPOTENTIALS
HYPERTENSION, 4663
BIOPSY
COLITIS, 4655*
CROHNS DISEASE, 5181
TECHNIQUES, 3803, 5181
ULCERATIVE COLITIS, 5181
BLEEDING
RADIOLOGY, 4212
SCANNING, SCINTILLATION, 4210
CROHNS DISEASE
COMPLICATIONS, 5179
DIAGNOSIS
COMPLICATIONS, 4667
POLYPS
FAMILIAL FACTORS, 4678
THERAPY
COMPLICATIONS, 4667

RECURRENCES
CROHNS DISEASE, 5171*

REFLUX
BILE
EROSIONS, 3939*
ULCER, 3929*
ESOPHAGUS
DIAGNOSIS, 4187*
GASTRECTOMY, 4403
HERNIA, 4314
HERNIA, HIATUS, 4363
RADIOLOGY, 4363
SPHINCTER, 4318
STENOSIS, 4335
SURGERY, 4304, 4306
GASTRECTOMY
SEQUELAE, 4403
PEPTIC ULCER
SURGERY, 4484*

REGIONAL ENTERITIS
SEE ALSO ENTERITIS
DIETARY FACTORS, 5167*
DUODENUM, 5169*
ETIOLOGY
GLUTEN, 5159*
IMMUNOGLOBULINS, 5159*
IMMUNOLOGY, 5184
RADIOLOGY, 5175

RESPIRATORY SYSTEM
CIRRHOSIS
DISEASES ASSOCIATED WITH, 4934
GASTROINTESTINAL
ENDOSCOPY, 4211
MARASMUS
DISEASES ASSOCIATED WITH, 5078

RESPIRATORY SYSTEM (continued)
PEPTIC ULCER
DISEASES ASSOCIATED WITH, 4533

REVIEWS
ANTRUM
ION TRANSPORT, 3842
BILE DUCT
PATHOLOGY, 5068
SURGERY, 5068
BILIRUBIN
METABOLISM, 4116, 4174
CELIAC DISEASE
ANEMIA, 4634
CHILDREN, 4632, 4642
IMMUNOGLOBULINS, 4640
IMMUNOLOGY, 4640
CHOLERA
ETIOLOGY, 5037
THERAPY, 5037
COLON
ABSORPTION, 3840
MOTILITY, 3908
SECRETION, 3840
CROHNS DISEASE, 4712, 5183
DEGLUTITION
MERYCISM, 4309
DERMATITIS HERPETIFORMIS, 4639
DIARRHEA
CHILDREN, 5020
DUODENUM
ENDOSCOPY, 4209
ECHINOCOCCOSIS, 5138
ENTEROCOLITIS, NECROTIZING, 5080
ESOPHAGUS
DYSKINESIA, 4305
ENDOSCOPY, 4209
GASTROINTESTINAL
ENDOSCOPY, 4203
HORMONE, 5039
GASTROINTESTINAL DISEASE
GASTRIN, 5024
GASTROSTOMY
TECHNIQUES, 4409
GLUTEN
MALABSORPTION, 4613
HELMINTHIASIS, 5134
HIRSCHSPRUNGS DISEASE
DIAGNOSIS, 4708
ETIOLOGY, 4708
PATHOLOGY, 4708
LIVER
NEOPLASMS, BENIGN, 4819
NEOPLASMS, MALIGNANT, 4783*, 4819
LIVER DISEASE
LIVER FUNCTION TESTS, 4292
RADIOLOGY, 4271
MALABSORPTION
DRUG-INDUCED, 4641
PANCREAS
PSEUDOCYST, 4769
PANCREAS DISEASE
ALCOHOL, 4767
ALCOHOLISM, 4730
PANCREATIC DUCT
PATHOLOGY, 5068
SURGERY, 5068

REVIEWS (continued)
PANCREATITIS
COMPLICATIONS, 4777
DIAGNOSIS, 4777
SURGERY, 4777
PARASITES AND PARASITIC DISEASE, 5133
PEPTIC ULCER, 4513
SURGERY, 4528
SALIVARY GLANDS
DISEASES, 5124
PATHOLOGY, 5125
ULTRASTRUCTURE, 5125
SCHISTOSOMIASIS
LIVER, 5144
SMALL INTESTINE
ABSORPTION, 3826
HEALING, 4595
ULTRASTRUCTURE, 3826
STOMACH
DYSKINESIA, 4305
ENDOSCOPY, 4209
MOTILITY, 3886
NEOPLASMS, MALIGNANT, 4468
ULCERATIVE COLITIS, 4712, 4713
UROBILINOGEN
EXCRETION, 4116
ZOLLINGER-ELLISON SYNDROME
DIAGNOSIS, 5115
ETIOLOGY, 5115

REYES SYNDROME
TRANSFUSION, 4784*

RISK FACTORS
HEPATITIS, NONVIRAL
DRUG-INDUCED, 4859*

RUPTURE
ESOPHAGUS
CONTRAST MEDIA, 4195*
ETIOLOGY, 4342
SURGERY, 4341, 4342
VARICES, 4938
GALLBLADDER
CYSTS, 4979
LIVER
ORAL CONTRACEPTIVES, 4790*
SMALL INTESTINE
HERNIA, 4583
NEOPLASMS, BENIGN, 4562
SALIVARY GLANDS
AMYLASE
SECRETION, 3911
BICARBONATE SECRETION, 3825
DISEASES
REVIEWS, 5124
ENZYMES
AGE FACTORS, 3913
GLYCOGEN
CATECHOLAMINES, 3912
MORPHOLOGY
PANCREATITIS, 3796
NEOPLASMS
CHILDREN, 5109, 5111
RADIOLOGY, 5109
SURGERY, 5111

SALIVARY GLANDS (continued)
NEOPLASMS, MALIGNANT, 5063
PATHOLOGY, 5101
ULTRASTRUCTURE, 5101
NUCLEIC ACIDS
SYNTHESIS, 3912
PATHOLOGY
REVIEWS, 5125
PROTEIN
FEEDING, 3914
SECRETION
TECHNIQUES, 5100
ULTRASTRUCTURE
REVIEWS, 5125

SALMONELLOSIS
CARRIER STATE
CHILDREN, 5072
DRUG TREATMENT, 5058
EPIDEMIOLOGY, 5030, 5044, 5046
INFECTION
IMMUNOGLOBULINS, 5107
IMMUNOLOGY, 5107
SMALL INTESTINE
PERFORATION, 5086

SARCOIDOSIS
STOMACH
DIAGNOSIS, 4476

SCANNING, SCINTILLATION
BILIARY DISEASE, 4250
ESOPHAGUS DISEASE
RADIOISOTOPES, 4200*
HEPATITIS, CHRONIC, 4911
INTESTINE
HETEROTOPIA, 4210
LIVER
NEOPLASMS, 4821
LIVER DISEASE, 4810
MECKELS DIVERTICULUM, 4186*
BLEEDING, 4545*
PANCREAS
TECHNIQUES, 4249
PANCREAS DISEASE, 4250
PAPILLA OF VATER
TECHNIQUES, 4248
RECTUM
BLEEDING, 4210

SCHISTOSOMIASIS
DRUG TREATMENT, 4935, 5150
COMPLICATIONS, 5132*
LIVER
COMPLICATIONS, 5145
DIAGNOSIS, 5145
REVIEWS, 5144

SCLERODERMA
ESOPHAGUS, 4339
DIAGNOSIS, 4337, 4338
RADIOLOGY, 4338
LIVER
PATHOLOGY, 4841
LIVER DISEASE
AUTOIMMUNITY, 4904*
LIVER FUNCTION TESTS, 4841

SECRETIN
BLOOD
AMINO ACIDS, 3937*
FATTY ACIDS, 3937*
PEPTIC ULCER, 4490*
SMOKING, 4490*
SUGAR, 3937*
SMALL INTESTINE
KINETICS, CELL, 4155*
STOMACH
MOTILITY, 3888
SECRETION, 3926*

SECRETION
AMYLASE
SALIVARY GLANDS, 3911
BICARBONATE
PANCREATITIS, 4754
BILE
BILIARY DISEASE, 4806
CHOLERETIC AGENTS, 4806
DRUG EFFECTS ON, 4967
LIVER DISEASE, 4806
NARCOTICS, 4132
VAGOTOMY, 4967
CHOLESTEROL
BILE ACIDS, 4009*
COLON
REVIEWS, 3840
DRUGS
INTESTINE, 3822
ENZYMES
PANCREAS, 4736
GASTRIN
PATHOLOGY, 4471
PEPTIC ULCER, 4471
STOMACH DISEASE, 4471
GASTROINTESTINAL
DRUG EFFECTS ON, 3918
GASTROSTOMY, 4416
INTUBATION, 4416
LIPIDS
BILE, 4941*
PHENOBARBITAL, 3994*
LIPOPROTEINS
LIVER DISEASE, ALCOHOLIC, 4915
LIVER
MOTILITY, 3987
OBSTRUCTION, 4853
PANCREAS
AGE FACTORS, 4773
ALCOHOL, 3982, 4767
ALCOHOLISM, 4766
AMINES, 3979*
ANTIBIOTICS, 3983
BILE, 3985
CALCIUM, 3986
CHOLECYSTOKININ, 3986
DRUG EFFECTS ON, 3983
H2 RECEPTOR ANTAGONISTS, 4483*
HORMONE CONTROL, 3952
HYPOXIA, 3978*
MOTILITY, 3987
NARCOTICS, 3980*
NERVOUS CONTROL, 3977*
PANCREATITIS, 4766
PANCREOZYMIN, 3986
PEPTIC ULCER, 4483*

SECRETION (continued)
PANCREAS (continued)
PROTEIN, 3976*, 3990
SYMPATHOMIMETIC AGENTS, 3979*
TRANSPLANTATION, 3984
VAGOTOMY, 3981*
PEPSIN
BILE ACIDS, 3935*
HORMONE CONTROL, 3944*, 3951, 3954
HORMONE EFFECTS ON, 3887
SALIVARY GLANDS
TECHNIQUES, 5100
STOMACH, 4219
ANTICHOLINERGIC AGENTS, 3957
ANTRECTOMY, 3928*, 4400
BARIUM, 3923*
CHOLINERGIC AGENTS, 3926*
ETHNIC FACTORS, 4495*
GASTRIN, 3960, 3961, 3968
GLYCOPROTEINS, 4443
H2 RECEPTOR ANTAGONISTS, 3922*, 4483*
HORMONE CONTROL, 3952, 4494*
HORMONE EFFECTS ON, 3920*, 3953
HYPERCALCEMIA, 3943*
IRON, 3948
PARATHYROID GLAND, 4378*
PENTAGASTRIN, 3955, 3957
PEPTIC ULCER, 4483*, 4494*, 4495*, 4499*, 4534
SECRETIN, 3926*
ULCERATIVE COLITIS, 4714
ZINC
PANCREAS DISEASE, 4735

SEROTONIN
BLOOD
PANCREATITIS, 4756
GASTROINTESTINAL TRACT
IRRADIATION, 4164*
ILEUM
MAGNESIUM, 3875*
MOTILITY, 3875*

SERUM
SEE BLOOD
LIVER
REGENERATION, 4105

SEX FACTORS
CHOLELITHIASIS
EPIDEMIOLOGY, 4960
LIVER
CYTOCHROMES, 4083
NUCLEIC ACIDS
SYNTHESIS, 4081
PANCREATITIS
EPIDEMIOLOGY, 4778
PROTEIN
SYNTHESIS, 4081
STEROID
EXCRETION, 3857
STOMACH
NEOPLASMS, MALIGNANT, 4453, 4460

SHIGELLOSIS
IMMUNITY
GENETIC FACTORS, 5075

SHOCK
SMALL INTESTINE
CIRCULATION, 4168*
STOMACH
ULCER, 4369*

SHORT BOWEL SYNDROME
ANOMALY, CONGENITAL
FAMILIAL FACTORS, 4547
DISEASES ASSOCIATED WITH, 4547
SURGERY, 4600

SHUNT, INTESTINAL
BILE ACIDS
ABSORPTION, 4952*
FATTY LIVER
ETIOLOGY, 4001*
LIVER
PATHOLOGY, 4795*

SIGMOID
SEE ALSO LARGE INTESTINE

SMALL INTESTINE
SEE ALSO DUODENUM, JEJUNUM, ILEUM
SEE ALSO INTESTINE
ABSORPTION
AGE FACTORS, 4615
ALCOHOL, 3812*
CELIAC DISEASE, 4637
ENTEROCOLITIS, 4597
FATTY ACIDS, 3849
PATHOLOGY, 4637
RADIOISOTOPES, 3838
REVIEWS, 3826
ACETYLCHOLINE
BINDING, 3894
ANOMALY, CONGENITAL
BLEEDING, 4235
DIAGNOSIS, 4235
FAMILIAL FACTORS, 4547
ATRESIA
DIAGNOSIS, 4546
THERAPY, 4546
BACTERIA
SPRUE, TROPICAL, 4604*
BIOPOTENTIALS
VAGOTOMY, 3868*
BLEEDING
RADIOLOGY, 4212
CARBOHYDRATE
ABSORPTION, 3829
CELIAC DISEASE
NEOPLASMS, MALIGNANT, 4636
CHOLINESTERASE
ABSORPTION, 4158
CIRCADIAN RHYTHM
ALKALOIDS, 4156
CIRCULATION
SHOCK, 4168*
COLON
CROHNS DISEASE, 5193
CRYOSURGERY
HEALING, 4408
DIGESTION
ENTEROCOLITIS, 4597
DISACCHARIDASE
ALCOHOL, 4151*

SMALL INTESTINE (continued)
DISACCHARIDASE (continued)
ANTINEOPLASTIC AGENTS, 4157
CIRRHOSIS, 5090
DEFICIENCY, 5098
MALABSORPTION, 5049
PROTEIN, 5090
DISACCHARIDE
ALCOHOLS, POLYHYDRIC, 4162
DRUG ABSORPTION
ALCOHOL, 3858
ANTINEOPLASTIC AGENTS, 3860
STARVATION, 3860
VITAMIN B2, 3834
DRUG TREATMENT
DRUG TOXICITY, 4182
ELECTROLYTE
CIRRHOSIS, 5053
ENTEROCOLITIS, 5053
GASTRITIS, 5053
HEPATITIS, CHRONIC, 5053
PANCREATITIS, CHRONIC, 5053
PEPTIC ULCER, 5053
ULCERATIVE COLITIS, 5053
ENDOSCOPY
CROHNS DISEASE, 5185
ENTEROKINASE
DEFICIENCY, 4540*
ENZYMES
AGE FACTORS, 4615
EROSIONS
BLEEDING, 4425
FATTY ACIDS
OXIDATION, 4154*
FISTULA
DIAGNOSIS, 4579
HYPERALIMENTATION, 4581
SURGERY, 4580, 4582, 4673
THERAPY, 4579
FRUCTOSE
METABOLISM, 4149*
GLUCOSE
ABSORPTION, 3839
GLYCOLYSIS
DIETARY FACTORS, 4161
SUGAR, 4161
GLYCOPROTEIN
CHOLERA, 5074
GROWTH
HORMONE, 3785*
HEALING
REVIEWS, 4595
HEMATOMA, 4567
OBSTRUCTION, 4569
HEPATITIS, CHRONIC
ANTIGEN, AUSTRALIA, 4897
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4897
ILEUS
THERAPY, 4572
IMMUNOGLOBULINS
CHOLERA, 5074
CROHNS DISEASE, 5166*
INTUSSUSCEPTION
RADIOLOGY, 4586
SURGERY, 4585
ION TRANSPORT
DIURETICS, 4152*
DRUG EFFECTS ON, 4152*

SMALL INTESTINE (continued)
KINETICS, CELL
ALKALOIDS, 4156
AUTORADIOGRAPHY, 3801
CIRCADIAN RHYTHM, 4155*
NEONATE, 3801
PENTAGASTRIN, 4155*
SECRETIN, 4155*
LIPIDS
METABOLISM, 4151*, 4154*
SYNTHESIS, 4150*
LYMPHANGIECTASIA
CHILDREN, 4544*
TRIGLYCERIDE, MEDIUM CHAIN, 4543*
LYMPHATICS
HYPERPLASIA, 4444
METAPLASIA
DISEASES ASSOCIATED WITH, 4360
METASTASES
THERAPY, 4561
MICROORGANISMS
ANTIBIOTICS, 4171
MONOAMINOXIDASE
ABSORPTION, 4158
MOTILITY
ANTISPASMODICS, 3896
ATROPINE, 3863*
CATECHOLAMINES, 3874*
CERULEIN, 3863*
ILEUS, 3906
NERVOUS CONTROL, 3900
NERVOUS SYSTEM, 3876*
PROSTAGLANDIN, 3881, 3899
SURGERY, 3902
VAGOTOMY, 3868*
NEOPLASMS
DIAGNOSIS, 4560
SURGERY, 4565
NEOPLASMS, BENIGN, 4563
ANEMIA, 4564
ENDOSCOPY, 4293
RUPTURE, 4562
NEOPLASMS, MALIGNANT, 4566
ANEMIA, 4601
CROHNS DISEASE, 5180
DIAGNOSIS, 4555
OBSTRUCTION, 4554
SURGERY, 4558, 4588
ULTRASTRUCTURE, 4554
OBSTRUCTION
CROHNS DISEASE, 5193
NEONATE, 4548
NERVOUS SYSTEM, 4596
SURGERY, 4573
PATHOLOGY, 4611*
BLIND LOOP SYNDROME, 4628
CHOLERA, 5102
MALABSORPTION, 4620, 4622, 4638
SPRUE, 4638
SPRUE, TROPICAL, 4638
WHIPPLES DISEASE, 4610*
PERFORATION
SALMONELLOSIS, 5086
SURGERY, 4542*
PHOSPHATASE, ALKALINE
DEFICIENCY, 5098
PROTEIN
ABSORPTION, 3832

SMALL INTESTINE (continued)
PROTEIN (continued)
IMMUNITY, 4607*
INTOLERANCE, 4606*
PSEUDOTUMOR
PATHOLOGY, 4541*
RUPTURE
HERNIA, 4583
SURGERY
COMPLICATIONS, 4593
LIVER DISEASE, 4780*
NUTRITION, 4177
OBESITY, 4593, 4594, 4780*
PANCREAS, 4177
PANCREAS FUNCTION TESTS, 4177
SEQUELAE, 4587, 4593, 4780*
TECHNIQUES, 4587
TRAUMA
HEALING, 4595
ULCER
SURGERY, 4478
ULTRASTRUCTURE
HEPATITIS, CHRONIC, 4897
REVIEWS, 3826
VITAMIN B12
ABSORPTION, 3812*
VOLVULUS
SURGERY, 4549

SMOKING
BLOOD
SECRETIN, 4490*
GASTRITIS
ETIOLOGY, 4375*

SOCIOECONOMIC FACTORS
PARASITES AND PARASITIC DISEASE, 5153

SODIUM
BLOOD
CIRRHOSIS, 4919*
DIURETICS, 4919*
METABOLISM
KINETICS, 3809*
PANCREAS, 3809*
TRANSPORT
BICARBONATE SECRETION, 4152*
CROHNS DISEASE, 5164*

SONOGRAPHY
SEE ECHOGRAPHY

SPASM
DEGLUTITION, 4310

SPECIES DIFFERENCES
BILE ACIDS
METABOLISM, 4013*

SPHINCTER
ESOPHAGUS
ANTRECTOMY, 4486*
CHOLINERGIC AGENTS, 4486*
ESOPHAGITIS, REFLUX, 4299*
HERNIA, 4318
HORMONE EFFECTS ON, 4318
PRESSURE STUDIES, 3873*, 3905, 4486*
REFLUX, 4318
VAGOTOMY, 4486*

SPRUE
COMPLICATIONS
DIAGNOSIS, 4624
GLUTEN, 4624
THERAPY, 4624
SMALL INTESTINE
PATHOLOGY, 4638

SPRUE, TROPICAL
BACTERIA
SMALL INTESTINE, 4604*
FOLIC ACID
METABOLISM, 4623
SMALL INTESTINE
PATHOLOGY, 4638

STARVATION
AMINES
METABOLISM, 4072
LIVER
FATTY ACIDS, 4046
MORPHOLOGY, 4851
TRIGLYCERIDE, 4046
ULTRASTRUCTURE, 4851
OBESITY
LIVER, 4851
PANCREAS
ENZYMES, 3989
PROTEIN
SYNTHESIS, 4088
SMALL INTESTINE
DRUG ABSORPTION, 3860

STASIS
LIVER
LYMPHATICS, 4104

STATISTICAL STUDY
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4877, 4893
STOMACH
NEOPLASMS, MALIGNANT, 4467

STEATOSIS
SEE FATTY LIVER

STENOSIS
AMPULLA OF VATER
SIMULATION, 4139
BILIARY
ANGIOGRAPHY, 4259
COLON
DISEASES ASSOCIATED WITH, 4664
PANCREATITIS, 4664
DUODENUM
DRUG TREATMENT, 4550
ETIOLOGY, 4550
SURGERY, 4550
ESOPHAGUS
ABSCESS, 4336
ANOMALY, CONGENITAL, 4358
ENDOSCOPY, 4218
NEONATE, 4336
RADIOLOGY, 4218
REFLUX, 4335
SURGERY, 4334
LIVER DISEASE
SURGERY, 4847

STENOSIS (continued)
PYLORUS
ANOMALY, CONGENITAL, 4469
DIAGNOSIS, 4470
DISEASES ASSOCIATED WITH, 4547
NEONATE, 4470
THERAPY, 4470

STEROID
BILE
SYNTHESIS, 3994*
BILIARY
EXCRETION, 3857
EXCRETION
SEX FACTORS, 3857
METABOLISM
LIVER, 4128
LIVER DISEASE, 4849
ULCER
STRESS, 4385*
URINE
PEPTIC ULCER, 4536

STOMACH
SEE ALSO ANTRUM, PYLORUS
ABSORPTION
ASPIRIN, 4391*
BIOPOTENTIALS, 3843
ACID SECRETION
ATROPINE, 3965, 4499*
CHILDREN, 4215
CHOLINERGIC AGENTS, 3956
DIETARY FACTORS, 3931*
DRUG EFFECTS ON, 3972, 4519
ENZYMES, 3947
FATTY ACIDS, 3930*
GASTRIN, 3956, 3973
H2 RECEPTOR ANTAGONISTS, 3972
HEIDENHAIN POUCH, 3946*
HISTAMINE, 3972, 4492*
HORMONE CONTROL, 3944*, 3945*, 3951, 3954, 4396
HYPNOSIS, 3941*
INTESTINE, 3946*
ION TRANSPORT, 3949
NERVOUS CONTROL, 4396
PEPTIC ULCER, 4488*, 4492*, 4503*, 4505*, 4511*, 4519
PROSTAGLANDIN, 3927*
TECHNIQUES, 4215
ACIDITY
ANTACIDS, 4414
ENDOSCOPY, 4397
PHYSICAL FACTORS, 3862*
TECHNIQUES, 4397
ADENYL CYCLASE
FEEDING, 3970
IRRADIATION, 3970
AMINO ACIDS
ASPIRIN, 4391*
METABOLISM, 3933*
ANOMALY, CONGENITAL
PYLORUS, 4480
ATROPHY
AGE FACTORS, 4373*, 4393
RADIOLOGY, 4373*
BIOPOTENTIALS
ANGIOTENSIN, 3884

STOMACH (continued)
BIOPOTENTIALS (continued)
ASPIRIN, 4376*
BARIUM, 3923*
BILE ACIDS, 4376*
VAGOTOMY, 3883, 4530
BLEEDING
ALCOHOL, 4424
DRUG-INDUCED, 4374*, 4424
GASTRITIS, 4421
PEPTIC ULCER, 4421
SIMULATION, 4438
SURGERY, 4421, 5085
THERAPY, 4184, 4420
CARCINOIDS
PATHOLOGY, 4472
CHOLINESTERASE
VAGOTOMY, 3971
CIRCULATION
DIETARY FACTORS, 3931*
CROHNS DISEASE
RADIOLOGY, 5168*
CRYOSURGERY
HEALING, 4408
DIGESTION, 4418
DRUG EFFECTS ON
ANTIINFLAMMATORY AGENTS, 4381*
ENDOSCOPY, 4381*
DYSKINESIA
REVIEWS, 4305
ELECTROLYTE
ATROPINE, 4499*
ELECTROPHYSIOLOGY
BLEEDING, 3925*
ENDOTOXIN, 3925*
EPINEPHRINE, 3925*
ENDOSCOPY, 4216
CROHNS DISEASE, 5185
MACROGLOBULINEMIA, 4473
PEPTIC ULCER, 4415
REVIEWS, 4209
EROSIONS, 4434
ASPIRIN, 4388*, 4391*
BLEEDING, 4425
ENDOSCOPY, 4435
SEQUELAE, 4433
STRESS, 3939*
FOREIGN BODIES
DRUG TREATMENT, 4417
ENZYMES, 4417
GASTRITIS, ATROPHIC
KINETICS, CELL, 4412
GLYCOPROTEINS
CARBENOXALONE, 4503*
ULCERATIVE COLITIS, 4714
GROWTH
HORMONE, 3785*
HETEROTOPIA
ENTEROCOLITIS, 4450
SURGERY, 4450
HISTAMINE
VASODILATORS, 3934*
IMMUNOGLOBULINS
CIRRHOSIS, 4429
GASTRITIS, ATROPHIC, 4429
INTUSSUSCEPTION
HEMATEMESIS, 4475
IRON
BINDING, 3948
DEFICIENCY, 4380*

STOMACH (continued)
ISCHEMIA
BIOPOTENTIALS, 3861*
JEJUNUM
FISTULA, 5122
KINETICS, CELL, 4412
GASTRIN, 3964
LIPASE
PEPTIC ULCER, 4505*
LIPIDS
DIAGNOSIS, 4413
ETIOLOGY, 4413
LYMPHATICS
HYPERPLASIA, 4444
MERCAPTANS
PEPTIC ULCER, 4512*
MORPHOLOGY
GASTRIN, 3798, 3799, 3960
HEPATITIS, VIRAL, 4889
NEOPLASMS, MALIGNANT, 4432
PEPTIC ULCER, 4432
MOTILITY
ACIDS, 3889
ANALGESICS, 4377*
ANGIOTENSIN, 3884
BRADYKININ, 3864*
CATECHOLAMINES, 3871*, 3874*
GASTRIN, 3888
HORMONE EFFECTS ON, 3887
NERVOUS CONTROL, 3864*
NERVOUS SYSTEM, 3885
PANCREAS DISEASE, 3882
PEPTIC ULCER, 4506*
PREGNANCY, 4377*
PROSTAGLANDIN, 3881
PYLOROPLASTY, 4526
REVIEWS, 3886
SECRETIN, 3888
VAGOTOMY, 3883, 4526
MYELOMA
DIAGNOSIS, 4290
RADIOLOGY, 4290
NEOPLASMS
DIAGNOSIS, 4452
SURGERY, 4452
NEOPLASMS, BENIGN
ENDOSCOPY, 4293, 4474
FISTULA, 4431
RADIOLOGY, 4474
NEOPLASMS, MALIGNANT, 4457
AGE FACTORS, 4393, 4453, 4460
ALCOHOLISM, 5094
BIOPSY, 4449
BLEEDING, 4461
CIRRHOSIS, 4927
CYTOLOGY, 4224, 4389*
DIAGNOSIS, 4445, 4451, 4454, 4464
DISEASES ASSOCIATED WITH, 4447
ENDOSCOPY, 4192*, 4196*, 4224, 4383*, 4448, 4449
ENVIRONMENTAL FACTORS, 4442
EPIDEMIOLOGY, 4460, 4467
ETIOLOGY, 4463
FIBRINOLYSIS, 4394
GASTRECTOMY, 4456
GLYCOPROTEINS, 4443
HETEROTOPIA, 4574
KINETICS, CELL, 4412

STOMACH (continued)
NEOPLASMS, MALIGNANT (continued)
LACTATE DEHYDROGENASE, 4428
METASTASES, 4461, 4465
MORPHOLOGY, 4463
PATHOLOGY, 4368*, 4446, 4465
PHOSPHATASE, ACID, 4367*
PRECANCER, 4458, 4459, 4466
PROGNOSIS, 4454, 4464
RADIOLOGY, 4223
RADIOTHERAPY, 4441
RECURRENCE, 4456
REVIEWS, 4468
SEX FACTORS, 4453, 4460
STATISTICAL STUDY, 4467
SURGERY, 4440, 4445, 4462
SURVIVAL, 4451
THERAPY, 4454, 4464
NUCLEIC ACIDS
ELECTROPHORESIS, 4173
OBSTRUCTION
ANOMALY, 4477
PYLORUS, 4477
PATHOLOGY
ACIDITY, 4386*
ANEMIA, 4380*
BILE ACIDS, 4386*
CIRRHOSIS, 5099
GASTRIN, 3798
HEPATITIS, CHRONIC, 5099
HYPERCAPNIA, 4366*
ISCHEMIA, 4386*
PEPTIC ULCER, 4489*, 4512*
PENTAGASTRIN
PEPTIC ULCER, 4505*
PENTOSE
OXIDATION, 3950
PEPSIN
ATROPINE, 4499*
BILE ACIDS, 3935*
ISOLATION, 3959
PEPTIC ULCER, 4505*, 4511*
PERMEABILITY
ACIDITY, 3844
ALCOHOL, 3938*
ANTIINFLAMMATORY AGENTS, 3938*
BILE ACIDS, 3938*
CIRCULATION, 3844
OXYGEN, 3844
PHOSPHODIESTERASE
FEEDING, 3970
IRRADIATION, 3970
POLYPS
ENDOSCOPY, 4222, 4371*
PRECANCER
DISACCHARIDASE, 4387*
GASTRITIS, 4422
PATHOLOGY, 4446
PHOSPHATASE, ALKALINE, 4387*
PRESSURE STUDIES
PHYSICAL FACTORS, 3862*
PROTEIN
ULCERATIVE COLITIS, 4714
PSEUDOTUMOR
CHOLESTEROL, 4455
RADIOLOGY
ANALGESICS, 4411
CONTRAST MEDIA, 4220
TECHNIQUES, 4204, 4225

STOMACH (continued)
REGENERATION
ANTRECTOMY, 4400
SARCOIDOSIS
DIAGNOSIS, 4476
SECRETION, 4219
ANTICHOLINERGIC AGENTS, 3957
ANTRECTOMY, 3928*, 4400
BARIUM, 3923*
CHOLINERGIC AGENTS, 3926*
ETHNIC FACTORS, 4495*
GASTRIN, 3960, 3961, 3968
GLYCOPROTEINS, 4443
H2 RECEPTOR ANTAGONISTS, 3922*, 4483*
HORMONE CONTROL, 3952, 4494*
HORMONE EFFECTS ON, 3920*, 3953
HYPERCALCEMIA, 3943*
IRON, 3948
PARATHYROID GLAND, 4378*
PENTAGASTRIN, 3955, 3957
PEPTIC ULCER, 4483*, 4494*, 4495*, 4499*, 4534
SECRETIN, 3926*
ULCERATIVE COLITIS, 4714
SURGERY
COMPLICATIONS, 4479
ULCER
AGE FACTORS, 4393
BLEEDING, 4369*
CARBENOXALONE, 4370*, 4503*
CIRCULATION, 3940*
DRUG-INDUCED, 3929*
FIBRINOLYSIS, 4394
SHOCK, 4369*
SIMULATION, 3940*
SURGERY, 4478
ULTRASTRUCTURE
GLYCOPROTEINS, 3788*
VARICES
BLEEDING, 4184
VOLVULUS
SURGERY, 4419

STOMACH DISEASE
DRUG TREATMENT
ANTACIDS, 4302
DYSPEPSIA
DRUG TREATMENT, 4392
GASTRIN
SECRETION, 4471
PROTEIN
HYDROLYSIS, 5027
TRAUMA
INTUBATION, 4436

STRANGULATION
ESOPHAGUS
HERNIA, 4362

STRESS
BILE
PEPTIC ULCER, 4539
BILE ACIDS
PEPTIC ULCER, 4539
ENTEROCOLITIS
SIMULATION, 5017*
LIVER
MITOSIS, 4144

STRESS (continued)
STOMACH
EROSIONS, 3939*
ULCER
BEHAVIOR, 3921*
BURNS, 4390*
DIETARY FACTORS, 3921*
HORMONE EFFECTS ON, 4384*
PORTACAVAL SHUNT, 4439
PREVENTION, 4385*
STEROID, 4385*
VAGOTOMY, 5054

STRICTURE
ANORECTUM
CROHNS DISEASE, 5163*
BILE DUCT
CHOLEDOCHOLITHIASIS, 4988
PANCREATITIS, 4988
SURGERY, 4946*
ESOPHAGITIS, REFLUX
COMPLICATIONS, 4327

SUGAR
ABSORPTION, 3814
GALLBLADDER, 3819*
BLOOD
SECRETIN, 3937*
SMALL INTESTINE
GLYCOLYSIS, 4161
TRANSPORT
BILE ACIDS, 3837

SULFATE
ABSORPTION
ILEUM, 3848
DIARRHEA
ETIOLOGY, 5097

SULFOBROMOPHTHALEIN
EXCRETION
BILE, 4120

SURFACTANTS
ABSORPTION
INTESTINE, 3823
EXCRETION
FECES, 3823
URINE, 3823

SYMPATHOMIMETIC AGENTS
PANCREAS
SECRETION, 3979*
SYPHILIS
HEPATITIS, NONVIRAL
DIAGNOSIS, 4860*

TELANGIECTASIA
GASTROINTESTINAL
BLEEDING, 5123

TEMPERATURE
ENTEROCOLITIS
SIMULATION, 5017*
INTEGUMENTARY SYSTEM
CHOLECYSTITIS, 4537
PEPTIC ULCER, 4537
LIVER
TRANSPLANTATION, 4030

THALASSEMIA
LIVER
BIOPSY, 4840
PATHOLOGY, 4839

THYROID
COMPLICATIONS
CHOLELITHIASIS, 4997
HORMONE
CROHNS DISEASE, 5160*, 5161*
ULCERATIVE COLITIS, 5160*, 5161*
IODINE
CROHNS DISEASE, 5165*
ULCERATIVE COLITIS, 5165*
LIVER
PATHOLOGY, 4850

TOLERANCE TEST
AMMONIA
LIVER, 4101
MALABSORPTION
DIAGNOSIS, 4190*

TORSION
GALLBLADDER
DIAGNOSIS, 4964
ETIOLOGY, 4964
OMENTUM
SURGERY, 5064

TRACE ELEMENT
FOOD
ABSORPTION, 3846
LIVER
NECROSIS, 4071

TRANQUILIZERS
DIARRHEA
DRUG-INDUCED, 5079
DRUG METABOLISM
LIVER DISEASE, 4793*
GASTROINTESTINAL DISEASE
PSYCHOLOGICAL FACTORS, 5077

TRANSFERRIN
CIRRHOSIS
DIAGNOSIS, 4914
LIVER DISEASE, ALCOHOLIC
DIAGNOSIS, 4914

TRANSFORMATION
LIVER
MORPHOLOGY, 3786*

TRANSFUSION
HEPATITIS, NONVIRAL
DRUG-INDUCED, 4864
HEPATITIS, VIRAL
TRANSMISSION, 4872*
LIVER DISEASE
BLEEDING, 4804
REYES SYNDROME, 4784*

TRANSPLANTATION
KIDNEY
COMPLICATIONS, 4684
HEPATITIS, VIRAL, 4786*
IMMUNOSUPPRESSION, 4498*

TRANSPLANTATION (continued)
KIDNEY (continued)
PANCREATITIS, 4779
PEPTIC ULCER, 4498*
SEQUELAE, 4498*, 4791*
LIVER
IMMUNOSUPPRESSION, 4027
LIVER COMA, 4028, 4031
MAGNESIUM, 4074
MORPHOLOGY, 4030
PERFUSION, 4028
SURVIVAL, 4024
TECHNIQUES, 4024, 4025, 4026, 4029, 4074
TEMPERATURE, 4030
PANCREAS
PANCREATITIS, 4740*
SECRETION, 3984

TRANSPORT
AMINO ACIDS
BILE ACIDS, 3837
AMMONIA
COLON, 4654*
BILIRUBIN
ALBUMIN, 4123
DRUG EFFECTS ON, 4123
ENZYMES, 4121
LIVER, 4117, 4119, 4121, 4123
PHOSPHOLIPID, 4119
CALCIUM
INTESTINE, 3853
VITAMIN D, 3853
CHLORIDE
BICARBONATE SECRETION, 4152*
GALLBLADDER, 3856
CHOLESTEROL
LIVER, 4045
ELECTROLYTE
COLON, 3841
DRUG EFFECTS ON, 3833
LAXATIVES, 3841
GLUCOSE
LIVER, 4093
LIVER
REGENERATION, 4146
SODIUM
BICARBONATE SECRETION, 4152*
CROHNS DISEASE, 5164*
SUGAR
BILE ACIDS, 3837
UREA
COLON, 4654*
WATER
COLON, 3841
LAXATIVES, 3841

TRAUMA
BILIARY
DIAGNOSIS, 4966
SURGERY, 4966
GALLBLADDER
CYSTS, 4979
IRRADIATION
ENDOTOXIN, 4180
IMMUNOSUPRESSION, 4180
INTESTINE, 4180, 4185
LIVER, 4180
PREVENTION, 4185

TRAUMA (continued)
LIVER
COMPLICATIONS, 4835
SURGERY, 4832, 4834, 4835
PANCREAS
ENZYMES, 3992
SURGERY, 4732
SMALL INTESTINE
HEALING, 4595
STOMACH DISEASE
INTUBATION, 4436

TREATMENT
SEE THERAPY

TRIGLYCERIDE, 3829
ABSORPTION
BILE ACIDS, 3859
LIVER
DIABETES, 4844
FATTY LIVER, 4844
STARVATION, 4046

TRIGLYCERIDE, MEDIUM CHAIN
SMALL INTESTINE
LYMPHANGIECTASIA, 4543*

TUBERCULOSIS
APPENDIX, 4705
INTESTINE, 4652*
LIVER
INFECTION, BACTERIA, 4792*

ULCER
ANUS
CROHNS DISEASE, 5192
BILE
REFLUX, 3929*
BLEEDING
CIRRHOSIS, 4927
CECUM
CYTOMEGALOVIRUS, 4684
COLON
DIAGNOSIS, 4659
DUODENUM
BIOPSY, 5051
CARBENOXALONE, 4503*
ESOPHAGUS
HERNIA, HIATUS, 4313
LARGE INTESTINE
DISEASES ASSOCIATED WITH, 4665
SMALL INTESTINE
SURGERY, 4478
STOMACH
AGE FACTORS, 4393
BLEEDING, 4369*
CARBENOXALONE, 4370*, 4503*
CIRCULATION, 3940*
DRUG-INDUCED, 3929*
FIBRINOLYSIS, 4394
SHOCK, 4369*
STIMULATION, 3940*
SURGERY, 4478
STRESS
BEHAVIOR, 3921*
BURNS, 4390*
DIETARY FACTORS, 3921*
HORMONE EFFECTS ON, 4384*
PORTACAVAL SHUNT, 4439

ULCER (continued)
STRESS (continued)
PREVENTION, 4385*
STEROID, 4385*
VAGOTOMY, 5054
SURGERY
BLEEDING, 4478
COMPLICATIONS, 4991

ULCERATIVE COLITIS
SEE ALSO COLITIS
ANTICHOLINERGIC AGENTS, 4711*
BIOPSY
RECTUM, 5181
CHILDREN
SURGERY, 4720
CIRCULATION
PATHOLOGY, 3805
RADIOLOGY, 3805
COMPLICATIONS
HERNIA, 4710*
CROHNS DISEASE
DIAGNOSIS, 5194
EPIDEMIOLOGY, 4713
IMMUNOLOGY, 5184
JEJUNUM
BIOPSY, 5162*
LARGE INTESTINE
MICROORGANISMS, 4716
LEUKOCYTES
IMMUNOLOGY, 4715
PERFORATION
PERITONITIS, 5071
REVIEWS, 4712, 4713
SMALL INTESTINE
ELECTROLYTE, 5053
STOMACH
GLYCOPROTEINS, 4714
PROTEIN, 4714
SECRETION, 4714
SURGERY, 4711*
COMPLICATIONS, 4721
SEQUELAE, 4719
TECHNIQUES, 4717, 4718
THYROID
HORMONE, 5160*, 5161*
IODINE, 5165*

ULCERATIVE ENTERITIS
SURGERY, 5110

ULTRASTRUCTURE
DUODENUM
ABSORPTION, 3791*
FASCIOLIASIS
CECUM, 5158
INTESTINE
EMBRYOLOGY, 3793*
JEJUNUM
CELIAC DISEASE, 4633
LIVER, 3789*
ADENYL CYCLASE, 3794*
COENZYMES, 4113
CORTICOSTEROIDS, 4065
DEHYDRATION, 4053
DRUG EFFECTS ON, 4065
DRUG METABOLISM, 3787*
ENZYMES, 3797

ULTRASTRUCTURE (continued)
LIVER (continued)
HEPATECTOMY, 4143
HEPATITIS, CHRONIC, 4908
HEPATITIS, VIRAL, 4892
HORMONE, 3794*
IRRADIATION, 3790*
LEAD, 4064
MITOCHONDRIA, 4054
STARVATION, 4851
SALIVARY GLANDS
NEOPLASMS, MALIGNANT, 5101
REVIEWS, 5125
SMALL INTESTINE
HEPATITIS, CHRONIC, 4897
NEOPLASMS, MALIGNANT, 4554
REVIEWS, 3826
STOMACH
GLYCOPROTEINS, 3788*

UREA
SYNTHESIS
ALCOHOL, 4050
CORTICOSTEROIDS, 4094
FEEDING, 4050
LIVER, 4060, 4094
TRANSPORT
COLON, 4654*

URINE
CATECHOLAMINES
PEPTIC ULCER, 4536
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 4896
OXALATE
CIRRHOSIS, 5041
GASTROINTESTINAL DISEASE, 5041
MALABSORPTION, 5041
STEROID
PEPTIC ULCER, 4536
SURFACTANTS
EXCRETION, 3823

UROBILINOGEN
EXCRETION
REVIEWS, 4116

UROGENITAL SYSTEM
BILE DUCT
OBSTRUCTION, 4006*
INTESTINE
PATHOLOGY, 4660

URTICARIA PIGMENTOSA
GASTROINTESTINAL
PATHOLOGY, 4395

VAGOTOMY
ANTRUM
MOTILITY, 3866*
BILE
SECRETION, 4967
BLOOD
GASTRIN, 3924*
COLON
ENZYMES, 3915
DUODENUM
BIOPOTENTIALS, 3883
ENZYMES, 3915
MOTILITY, 3883

VAGOTOMY (continued)
ESOPHAGUS
SPHINCTER, 4486*
PANCREAS
SECRETION, 3981*
PEPTIC ULCER, 4508*, 4510*, 4521
ANTRECTOMY, 4525
COMPLICATIONS, 4592
ENTERITIS, 4592
JEJUNUM, 4522
PYLOROPLASTY, 4527
SEQUELAE, 4529
SURVIVAL, 4529
TECHNIQUES, 4523, 4524
SEQUELAE
ENTERITIS, 4402
SMALL INTESTINE
BIOPOTENTIALS, 3868*
MOTILITY, 3868*
STOMACH
BIOPOTENTIALS, 3883, 4530
CHOLINESTERASE, 3971
MOTILITY, 3883, 4526
ULCER
STRESS, 5054

VARICES
ESOPHAGUS
BLEEDING, 4184, 4308, 4938
PSEUDOTUMOR, 4359
RUPTURE, 4938
STOMACH
BLEEDING, 4184

VASODILATORS
STOMACH
HISTAMINE, 3934*

VASOPRESSIN
GASTROINTESTINAL
BLEEDING, 5015*
LIVER DISEASE
BLEEDING, 4804
PORTAL HYPERTENSION, 4918*

VIRUS
ENTERITIS
IMMUNOGLOBULINS, 5014*
FECES, 4172

VITAMIN A
AMINO ACIDS
METABOLISM, 4090
BLOOD
CIRRHOSIS, 4262
CROHNS DISEASE, 4262
MALABSORPTION, 4262
DEFICIENCY
COENZYMES, 4113
LIVER, 4090
LIVER
LIPIDS, 4048
METABOLISM
CIRRHOSIS, 4618
CROHNS DISEASE, 4618
MALABSORPTION, 4618
PANCREAS DISEASE, 4618

VITAMIN B1
DEFICIENCY
ABSORPTION, 3810*
LIVER
DEHYDROGENASE, 4086

VITAMIN B2
DEFICIENCY
ABSORPTION, 3810*
DRUG ABSORPTION
SMALL INTESTINE, 3834

VITAMIN B6
AMINO ACIDS
METABOLISM, 4091
BLOOD
PEPTIC ULCER, 4482
HISTAMINE
METABOLISM, 4159
METABOLISM
ALCOHOL, 4012*

VITAMIN B12
ABSORPTION
ALCOHOL, 3812*
SMALL INTESTINE, 3812*
BLOOD
HEPATITIS, VIRAL, 4883
CROHNS DISEASE
DRUG TREATMENT, 5195
HEPATITIS, VIRAL
RADIOISOTOPES, 4883

VITAMIN C
CHOLECYSTITIS
SIMULATION, 4134
PEPTIC ULCER
DRUG-INDUCED, 4500*

VITAMIN D
ABSORPTION
KIDNEY DISEASE, 5067
CALCIUM
ABSORPTION, 3847
TRANSPORT, 3853
CROHNS DISEASE
MALABSORPTION, 5195
DEFICIENCY
PROTEIN, 4112
GLYCOGEN
SYNTHESIS, 4112
INTESTINE
METABOLISM, 4163
METABOLISM
KIDNEY DISEASE, 5067
NUCLEIC ACIDS
METABOLISM, 4163
PROTEIN
BINDING, 4163
SYNTHESIS, 4112

VITAMIN E
AMINO ACIDS
METABOLISM, 4078
DEFICIENCY
COENZYMES, 4113

VITAMIN K
CLOTTING
LIVER, 4008*

VOLVULUS
SMALL INTESTINE
SURGERY, 4549
STOMACH
SURGERY, 4419

VOMITING
GASTROINTESTINAL
MOTILITY, 3878

WATER
ABSORPTION
CELIAC DISEASE, 4605*
GASTRIN, 3839
JEJUNUM, 3815*
TRANSPORT
COLON, 3841
LAXATIVES, 3841

WHIPPLES DISEASE
COMPLICATIONS
ETIOLOGY, 4626
THERAPY, 4626
DISEASES ASSOCIATED WITH
CHOLELITHIASIS, 4625
IMMUNOLOGY, 4611*
JEJUNUM
PATHOLOGY, 4625
PANCREAS
HETEROTOPIA, 4625
SMALL INTESTINE
PATHOLOGY, 4610*

WILSONS DISEASE
COPPER
KINETICS, 4782*
LIVER
CHILDREN, 4785*

XYLOSE
CIRRHOSIS
ABSORPTION, 4930
DIGESTION, 4930
GLUTEN
INTOLERANCE, 4602*
INTESTINE
ABSORPTION, 4231

YERSINIA
ENTEROCOLITIS, 5119, 5127

ZINC
ACRODERMATITIS ENTEROPATHICA, 5128
DRUG TREATMENT, 5113
DEFICIENCY
ACRODERMATITIS ENTEROPATHICA, 5129
DRUG METABOLISM
LIVER, 4057
IMMUNOLOGY, 5129
METABOLISM
ANTIBIOTICS, 4069
LIVER, 4069
SECRETION
PANCREAS DISEASE, 4735

ZOLLINGER-ELLISON SYNDROME
DIAGNOSIS, 5093
REVIEWS, 5115
DIARRHEA
HYPOCHLORHYDRIA, 5121
ETIOLOGY
REVIEWS, 5115
H2 RECEPTOR ANTAGONIST, 5025
PANCREAS
NEOPLASMS, 5116
THERAPY, 5093

*U.S. GOVERNMENT PRINTING OFFICE: 1975—211-007:4

DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MD. 20014

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE $300

CORRESPONDENCE CONCERNING THIS PUBLICATION

PAID SUBSCRIPTIONS: If your address has changed and you wish to continue receiving this publication, please check here ..... , enclose address label from this publication and your new address, and send to:

SUPERINTENDENT OF DOCUMENTS
U.S. GOVERNMENT PRINTING OFFICE
WASHINGTON, D.C. 20402

FREE SUBSCRIPTIONS: Contractors, Grantees, and Medical Libraries — If your address has changed and you wish to continue receiving this publication, please check here ..... , enclose address label from this publication and your new address, and send to:

SCIENTIFIC COMMUNICATIONS OFFICE
NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM,
AND DIGESTIVE DISEASES
NATIONAL INSTITUTES OF HEALTH
BUILDING 31, ROOM 9A-03
BETHESDA, MARYLAND 20014

Individual copies should be purchased from the Superintendent of Documents.

DHEW Publication No. (NIH) 76-66